THE CONCORDANCE BETWEEN DIABETIC NEPHROPATHY AND RETINOPATHY IN TYPE 2 DIABETES MELLITUS

Dissertation submitted in partial fulfilment of

the requirements for the degree of

D.M. (NEPHROLOGY)

BRANCH – III

DEPARTMENT OF NEPHROLOGY,

MADRAS MEDICAL COLLEGE,

CHENNAI – 600 003.

THE TAMIL NADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

AUGUST 2014

CERTIFICATE

This is to certify that this Dissertation entitled **"The concordance between diabetic nephropathy and retinopathy in type 2 diabetes mellitus"** is the bonafide original work done by Dr.Andrew Deepak Rajiv, under our guidance and supervision in the Department of Nephrology, Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai submitted as partial fulfillment for the requirement of D.M., (Nephrology) examination Branch III Nephrology, August 2014 of the Tamilnadu Dr.M.G.R Medical University Chennai.

DR. N.GOPALAKRISHNAN MD D.M. FRCP,
Professor and Head
Department of Nephrology
Madras Medical College,
Chennai - 3

Prof. R. VIMALA, MD
Dean,
Madras Medical College,
Chennai – 3

DECLARATION

I, Dr Andrew Deepak Rajiv , solemnly declare that the dissertation titled **"The concordance between diabetic nephropathy and retinopathy in type 2 diabetes mellitus"** is the bonafide work done by me at Department of Nephrology, Madras Medical College under the expert guidance and supervision of **Dr.N.GOPALAKRISHNAN D.M, FRCP,** Professor of Nephrology, Madras Medical College. The dissertation is submitted to the Tamilnadu Dr.M.G.R Medical University towards partial fulfillment of requirement for the award of D.M. Degree (Branch III) in Nephrology.

Postgraduate student,
Dept. of Nephrology,
Madras Medical College,
Chennai

Place : Chennai

Date :

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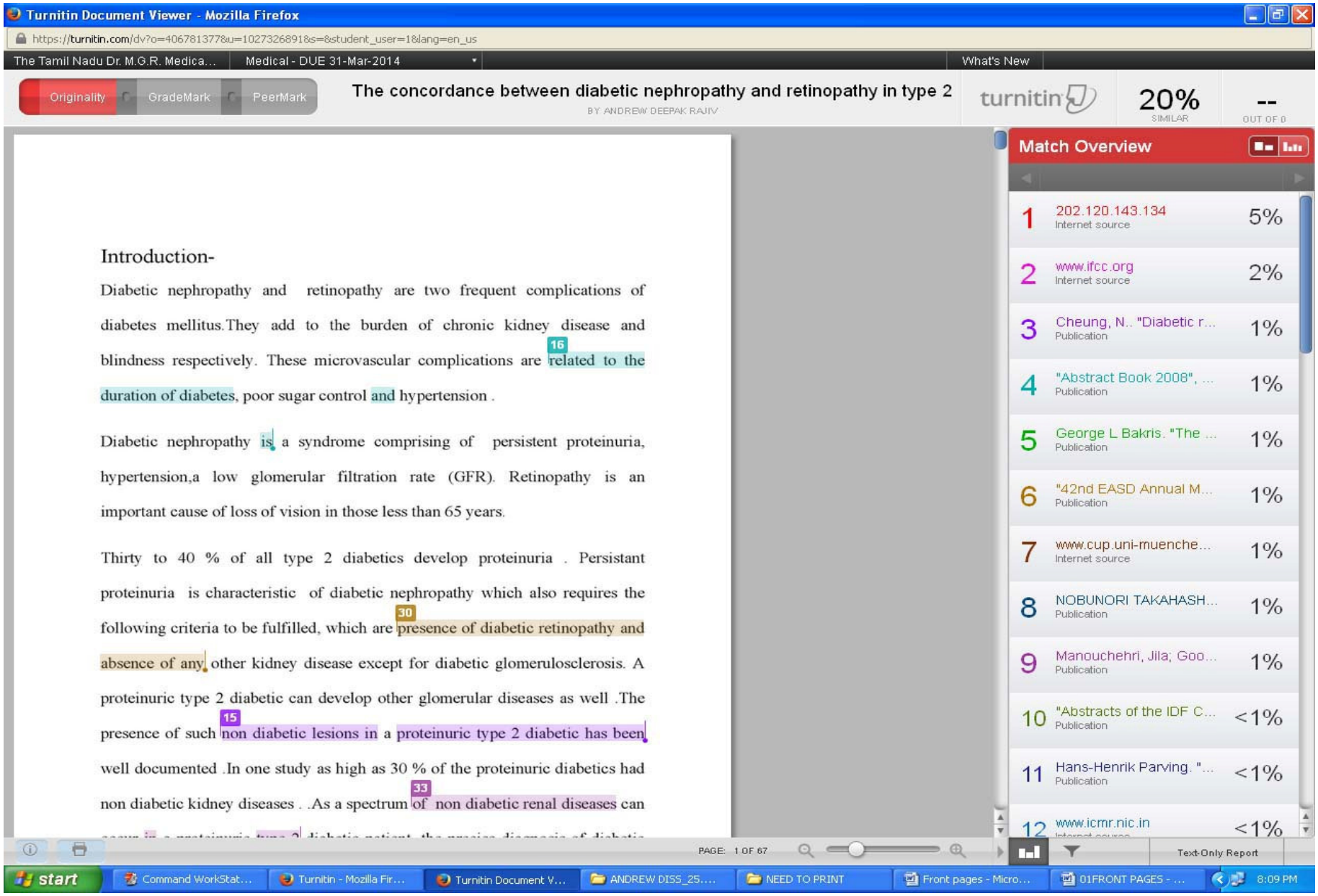
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Introduction-
Diabetic nephropathy and retinopathy are two frequent complications of diabetes mellitus.They add to the burden of chronic kidney disease and blindness respectively. These microvascular complications are related to the duration of diabetes, poor sugar control and hypertension .
Diabetic nephropathy is a syndrome comprising of persistent proteinuria, hypertension,a low glomerular filtration rate (GFR). Retinopathy is an important cause of loss of vision in those less than 65 years.
Thirty to 40 % of all type 2 diabetics develop proteinuria . Persistant proteinuria is characteristic of diabetic nephropathy which also requires the following criteria to be fulfilled, which are presence of diabetic retinopathy and absence of any other kidney disease except for diabetic glomerulosclerosis. A proteinuric type 2 diabetic can develop other glomerular diseases as well .The presence of such non diabetic lesions in a proteinuric type 2 diabetic has been well documented .In one study as high as 30 % of the proteinuric diabetics had non diabetic kidney diseases . .As a spectrum of non diabetic renal diseases can
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CERTIFICATE OF APPROVAL

To
Dr. Andrew Deepak Rajiv,
Postgraduate,
Department of Nephrology
Madras Medical College &
Rajiv Gandhi Government General Hospital,
Chennai-3.

Dear **Dr. Andrew Deepak Rajiv,**

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"To study the Concordance between Diabetic Nephropathy and Retinopathy in Type-2 Diabetes Mellitus"** No.20122012.

The following members of Ethics Committee were present in the meeting held on 11.12.2012 conducted at Madras Medical College, Chennai-3.

1. Dr. G. Sivakumar, MS FICS FAIS	-- Chairperson
2. Prof. B. Kalaiselvi, MD Vice Principal, MMC, Ch-3	-- Member Secretary
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5. Thiru. S. Govindasamy, BA, BL	-- Lawyer
6. Tmt. Arnold Saulina, MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

11/12/12

Member Secretary, Ethics Committee

MEMBER SECRETARY
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CONTENTS

INTRODUCTION

Diabetic nephropathy and retinopathy are two frequent complications of diabetes mellitus.[1]They add to the burden of chronic kidney disease and blindness respectively.[1] These microvascular complications are related to the duration of diabetes, poor sugar control and hypertension .

Diabetic nephropathy is a syndrome comprising of persistent proteinuria, hypertension,a low glomerular filtration rate (GFR).[1] Retinopathy is an important cause of loss of vision in those less than 65 years.[1]

Thirty to 40 % of all type 2 diabetics develop proteinuria .[2] Persistant proteinuria is characteristic of diabetic nephropathy which also requires the following criteria to be fulfilled, which are presence of diabetic retinopathy and absence of any other kidney disease except for diabetic glomerulosclerosis.[2] A proteinuric type 2 diabetic can develop other glomerular diseases as well .[3]The presence of such non diabetic lesions in a proteinuric type 2 diabetic has been well documented .In one study as high as 30 % of the proteinuric diabetics had non diabetic kidney diseases .[3] .As a spectrum of non diabetic renal diseases can occur in a proteinuric type 2 diabetic patient ,the precise diagnosis of diabetic nephropathy requires histological evaluation by renal biopsy.[3]

While there is a strong concordance between diabetic nephropathy and retinopathy, in type 1 diabetes mellitus ,in type 2 diabetes the concordance is around 60 % as per studies done by Parving et al[4] . Studies by J Prakash et al showed diabetic retinopathy not to be a good predictor of diabetic nephropathy[2]. In 50 % of those without retinopathy ,nephropathy was noted. Non diabetic renal lesions alone or coexisting with DN were noted in 40 % of those with retinopathy[2] .

Hence we felt the need of a study to determine the concordance between diabetic nephropathy and retinopathy and to see the spectrum of non diabetic kidney diseases in proteinuric type 2 diabetics of our department .

AIMS AND OBJECTIVES

- To study the concordance between diabetic nephropathy and retinopathy in patients of type 2 diabetes mellitus.

- To study the clinical and lab profile of a patient with diabetic nephropathy Vs a patient with non diabetic nephropathy.

- The spectrum of non diabetic renal disease in a proteinuric type 2 diabetic .

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REVIEW OF LITERATURE

The prevalence of diabetes is high among Indians so studies on diabetic complications are important .[3]

Diabetic nephropathy and retinopathy are two frequent complications of diabetes mellitus.[1]They progress to chronic kidney disease and loss of vision ,causing significant burden on the patient and health system. Diabetic nephropathy is a syndrome comprising of persistent proteinuria, hypertension and a low glomerular filtration rate (GFR).[1] There is a high risk of cardiac morbidity and mortality. This is seen in approximately 40% of type 2 diabetics.[1]

Retinopathy is an important complication of diabetes mellitus .It is a leading cause of blindness in adults.[1] Diabetic retinopathy is primarily classified into non proliferative DR (NPDR) and proliferative DR (PDR).[1] Visual disturbance is in diabetic retinopathy is due to macular edema and PDR. Vision loss can also occur due to vitreous haemorrhage from the new vessels in PDR.[1]

Overview of Diabetic nephropathy

Historical facts –

Kimmelstiel and Wilson described the renal pathology of DN by autopsy as early as 1936. Eight cases were found to have the typical nodular glomerulosclerosis of diabetic nephropathy. These findings were confirmed by others also .Diffuse glomerulosclerosis was described and differentiated from the nodular forms.[5-6]

Later studies involving percutaneous renal biopy and electron microscopy showed the earliest changes of diabetic nephropathy to be thickening of the basement membrane(GBM) and mesangial expansion.[6]

Epidemiology

DN is well studied in patients with type 1 diabetes. The onset of disease is usually known in them.25% to 45% of these patients develop nephropathy in their lifespan.[7] The peak time to develop nephropathy is 10 to 15 years from onset. In patients with type 2 diabetes, the prevalence of nephropathy is reported to be lower. Nephropathy developed in 50% of type 2 diabetic Pima Indians.[8] This was 20 years after diagnosis and 15% progressed to ESRD. Proteinuria is known to predispose for cardiovascular disease. It is thought that earlier studies underestimated the prevalence of DN. The actual prevalence of DN is difficult to determine ,as early cardiovascular mortality would have preceded the development of ESRD.

Recent data shows the risk of nephropathy to be same in both types of diabetes . The time to develop proteinuria from the onset and to progress to ESRD once proteinuria develops, were same in types 1 and 2 diabetes.[9]

Natural history of Diabetic nephropathy –

Mogensen et al proposed five stages for renal involvement.[10]

Stage 1: glomerular hyperfiltration.

Even with optimal blood glucose, GFR remains above normal in 25% to 40% of patients. In this group fall in GFR developed at a faster rate,compared to controls with normal GFR. This raised GFR is due to increased glomerular capillary filtration surface. Higher GFR early in the course of disease makes it more likely to develop DN.[11]

Stage 2: Early lesions

Mild thickening of glomerular basement membrane takes place 18 to 24 months from the start of type 1 diabetes.[10] Glycosylation of the basement membrane causes an increase in filtration of proteins.The negative charge of the basement membrane is reduced which causes increased loss of albumin as it is no longer repelled by basement membrane.[11]

Stage 3: incipient diabetic nephropathy—stage of microalbuminuria.

Microalbuminuria is the urine albumin excretion of 20 to 200 g/min per day. It is associated with loss of renal function and poor outcomes . Associated with vascular injury in other organs also.[10]

Stage 4: clinical nephropathy—macroalbuminuria, decline in GFR .

Usually manifests in patients who have diabetes for 15 to 20 years. Without intervention, the GFR in these patients, falls at about 1 mL/min/month.

Stage 5: End-stage renal disease-

ESRD occurs after 20 to 30 years of diabetes. This is seen in 30% to 40% with type 1 diabetes. Uremic symptoms and signs are seen at much higher creatinine clearances than nondiabetics.[11]

Pathology -

The 3 main pathological changes in the glomeruli of DN:

(1) mesangial expansion.

(2) glomerular basement membrane(GBM) thickening.

(3) glomerular sclerosis.[12]

Glomerular sclerosis with a nodular appearance is called the ‘‘Kimmelstiel-Wilson lesion’’. Hyaline deposits in the glomerular arterioles

reflects the leakage of proteins. These are namely fibrin, immunoglobulins, and complement .[13]

The mesangial expansion and glomerulosclerosis do not always develop together .This means that they have different pathogenetic mechanisms.[12] Mesangial expansion is caused by hyperglycemia which causes an increased formation or glycosylation of mesangial proteins.[13]

Pathogenesis of Diabetic nephropathy-

Factors involved -

- Hemodynamic pathways
- Hyperglycemia and Advanced glcosylation end products
- Protein kinase C pathways
- Aldose reductase pathways
- Prorenin
- Cytokines
- Oxidative stress
- Genetic susceptibility

Hemodynamic factors –

Glomerular hyperfiltration is because of decreased resistance in both the afferent and efferent arterioles.The afferent arteriole has lesser resistance than the efferent.[12] Factors involved in this defective autoregulation are

prostanoids,nitric oxide,vascular endothelial growth factor VEGF. TGF-β1, and the renin–angiotensin system mainly angiotensin II are the other factors mainly seen.[13] The hemodynamic factors result in albumin to leak from the glomerular capillaries. Increased production of mesangial cells, thickening of the GBM and injury to podocytes are due to hemodynamic factors. Glomerular hypertension and hyperfiltration play an important role in the pathogenesis diabetic nephropathy as use of angiotensin blockers prevents loss of nephrons. Blocking the angiotensin system prevents the fibrosis inducing effects of angiotensin II on TGF -β.[15]

Hyperglycemia and advanced glycosylation end products

Hyperglycemia causes an increase in mesangial cell proliferation and hypertrophy.[16] Hyperglycemia is associated with an increased matrix production and basement membrane thickening. Mesangial cell expansion is due to an increase in the mesangial cell glucose concentration.[17] Hyperglycemia causes the upregulation of VEGF expression in podocytes.This markedly increases vascular permeability.[18]

Advanced glycosylation end products

Glycosylation of proteins is one of the causes of nephropathy. Long standing increased sugars, causes glucose to combine with free amino acids on tissues.[17] This nonenzymatic process has an affect on the glomerular basement membrane[18].Other matrix components in the glomerulus are also

affected.The early formation of reversible glycation products are replaced later by irreversible advanced glycation products. The levels of AGE are increased in the diabetics. This is more in renal failure,as it is excreted in the urine.[18] This leads to accumulation of advanced glycosylation end products which has a tendency to cross-link with collagen. This contributes to development of DN.

Protein kinase C

Hyperglycemia causes diabetic nephropathy by activation of PKC. Activation of this enzyme causes the increased production of prostanoids which cause vasodilatation.This contributes to glomerular hyperfiltration. PKC activation causes the formation of diacylglycerol which leads to oxidative stress.[19] PKC activation also increases the activity of mitogen-activated protein kinases (MAPK).

Aldose reductase pathway

This enzyme helps in changing sugars into alcohols. By this enzyme glucose is converted into sorbitol and galactose into galactitol.These alcohols ,fail to diffuse out from the cells .Their accumulation intracellularly causes osmosis thereby allowing entry of water into the cell.[20]This results in electrolyte imbalances and also the depletion of myoinositol. This causes tissue damage to occur.[21]

Prorenin

Increased prorenin activity in the plasma was found to be a risk factor for the development of diabetic nephropathy.[21] The prorenin receptors are located in the mesangium and podocytes of the kidney. Prorenin binding to specific tissue receptors promotes the activation of p44/p42 MAPK.[22] The blocking of Prorenin receptors prevented MAPK activation which blocked nephropathy from progressing .

Cytokines

Activation of cytokines, enzymes causing profibrosis, inflammation and VEGF are some of the factors involved in increased matrix formation in diabetic nephropathy.[23] Hyperglycemia increases VEGF expression acts to cause endothelial injury in diabetes.[22]

The VEGF and angiopoetin cause vessel leakage of proteins .These two factors play important roles in development of retinopathy and nephropathy. VEGF stimulates the formation of α3 chain of collagen which is an important component of the GBM. It is seen that increased formation of collagen leads to the thickening of the GBM.[22]

Some studies however do not show VEGF as a causative factor , Baelde *et al* demonstrated VEGF mRNA levels,to be low in the glomeruli

for some cases of diabetic nephropathy.This was associated with reductions in podocytes number and development of nephropathy.[24]

Hyperglycemia can increase the production of TGF-β1 in the glomeruli.[21]

Inflammatory cytokines are responsible for the progression of diabetic nephropathy.They are interleukins 1,6,8 (IL‑1,6,8) and tumor necrosis factor.Increased levels of interleukins correlate well with development of nephropathy. IL-1 acts on chemotactic factors and adhesion molecules altering their expression. It also causes intraglomerular hypertension. Increases vascular endothelial permeability. It also has an effect to increase hyaluron production by renal tubular epithelial cells.[22] IL-6 acts likely by increasing the glomerular basement membrane thickening. It also has a possible role to increase endothelial permeability and mesangial proliferation. IL-18 increases the production of other inflammatory cytokines.

Oxidative stress

Reactive oxygen species are produced in the nephrons during cellular metabolism. These molecules are countered by a large number of antioxidants and free radical scavengers. Reactive oxygen species have negative effects. They cause peroxidation of cell membranes lipids.They

also cause renal vasoconstriction. Hyperglycemia shifts the balance towards formation of reactive oxygen species.Most of these are generated in the mitochondria.[21].Concentrations of the reactive oxygen species are higher in those with more severe nephropathy.

Genetic susceptibility

As mentioned earlier increased production of angiotensin II determines the initiation and progression of DN. Angiotensin II mainly acts by affecting hemodynamic and nonhemodynamic mechanisms.[24] Polymorphism of the ACE gene due to inversion or deletion (ACE/ID) occurs. This is associated with varying levels of circulating ACE. The probability of developing DN is more for patients,having a sibling or parent with history of DN. Patients with DD polymorphism of the ACE gene have a high risk of developing diabetic nephropathy and progressive renal failure.

Clinical predictors and risk factors for diabetic nephropathy and Retinopathy

1)Glycemic control

Diabetic nephropathy develops more often in patients with long standing uncontrolled hyperglycemia. Diabetics with a hemoglobin A1c level less than 8.1% are at much lower risk for DN[25].Randomized clinical trials have confirmed this association both for nephropathy and retinopathy.

The United Kingdom Prospective Diabetes Study (UKPDS)in type 2 diabetics,showed fewer patients of the tight blood sugar control to progress to microalbuminuria. Less in the intensive blood sugar group compared to conventional group progressed to microalbuminuria (27% versus 39%) and proteinuria (7% versus 13%)over a 15 year followup period[26]. The Diabetes Control and Complications Trial (DCCT) highlighted the benefits of intensive therapy for glycemic control. They showed a reduction in microalbuminuria by 39% and albuminuria by 54% respectively[27].The DCCT showed that in the intensive arm there was a 76% reduction in the development of retinopathy[28].Even in those with established retinopathy there was a 80% reduction in progression,compared to those with conventional control. These targets were achieved by maintaining a median hemoglobin A1c level of 9.1% and 7.3 % in the conventional and intensive treatment group respectively[28].These benefits persisted even after 7 years of continued follow-up. This finding was demonstrated by Epidemiology of Diabetes Interventions and Complications study. The difference in HbA1c levels between two groups was 0.4% .Though this diference appears small it still provided benefit I year after completion of the DCCT. Wisconsin study of DR was a population based study. This study was done to determine the prevalence and severity of DR in their study population. The study concluded that there is a strong association between severity of retinopathy and high levels of HBA1C.[27]

For patients with already existing advanced retinopathy, strict control of blood glucose will not be helpful in preventing the progression of retinopathy.[27]

2)Duration of disease

The total duration of disease was found to be an important risk factor for the development and progression of retinopathy[26].The Wisconsin epidemiologic study of retinopathy, showed that the longer the duration of diabetes more the prevalence of retinopathy in the study population .[27]

3) Blood pressure -

Studies show increased systolic blood pressure to be a strong risk factor for both diabetic nephropathy and retinopathy. This risk was more for retinopathy[26]. In UKPDS trial it was seen that good blood pressure control led to decreased progression of retinopathy. A 10/5 reduction of blood pressure caused a 34% reduction in progression of retinopathy and 47% decreased risk of deterioration in visual acuity.[27]

4)Glomerular filtration rate –

Patients with glomerular hyperfiltration had an increased risk for developing diabetic nephropathy .The glomerular hyperfiltration and hypertrophy are due to intraglomerular hypertension and capillary wall stress.[29]

In type 2 diabetes , 45% have a GFR that is significantly more than age matched nondiabetics and obese controls The amount of hyperfiltration is lesser in type 2 diabetics compared to type 1. Type 2 diabetics being older are more likely to have atherosclerotic vascular disease,this factor probably limits the increase in glomerular filtration.[30]

Diabetic Retinopathy

Natural history and classification

In the early stages of diabetic retinopathy there is loss of pericytes near the capillaries of the retina. This causes weakness in the walls of the capillaries.[31] Microaneurysm develop as a result of this weakness.The increased permeability of the capillaries cause them to leak fluid . This capillary wall weakness, causes impaired function of the blood vessels ,this gradually leads to areas of ischemia and infarction.[32] These changes cause local growth factors to be produced leading to new vessel proliferation.

Classification of diabetic retinopathy

1) Non proliferative diabetic retinopathy- characterised by microaneurysms,hemorrhages and exudates .This does not affect vision.[33]
2) Retinopathy with maculopathy- characterised by macular edema causing vision to be affected .

3) Proliferative diabetic retinopathy – characterised by neovascularisation ,fibrous proliferation and vitreous haemorrhages which can lead to sudden loss of vision.[34]
4) Advanced diabetic eye disease –which leads to vitreous opacities,hemorrhages and retinal detachment .

Pathology and pathogenesis of diabetic retinopathy

In nonproliferative DR the main features are structural abnormalities of the retinal vessels. Mainly the capillaries are involved, although venules and arterioles are also frequently involved. This causes retinal ischemia, edema and haemorrhages.[33] Proliferative DR has the previously mentioned changes, in addition features like optic disc, retinal or iris neovascularization. Retinal neovascularization e vitreous haemorrhages. Vascular endothelial growth factor (VEGF) is a powerful stimulator of vasculogenesis and mitogenesis.[33] Increased VEGF levels are present in the retina of diabetic patients.[34] As mentioned previously they act by increasing the vessel permeability ,causing increased amounts of vascular leakage.[34]

Apoptosis of retinal capillary pericytes and, to a smaller extent the retinal capillary endothelial cells occur.This has been shown in early DR[35]. Pericyte glutathione defends against peroxidation. High glucose states lead to their depletion[36]. This high concentration of glucose serves as an

apoptotic signal for retinal pericytes. The apoptosis that occurs leads to a series of events that first result in NP DR. As the loss of pericytes and damage to endothelial cells progresses, there is a release of transforming growth factor [TGF]-b) . As mentioned earlier, aldose reductase which acts by converting sugars into their respective alcohols, is found in high concentrations in retinal pericytes causing damage to it.[36]

Relation between diabetic nephropathy and retinopathy –

Patients with type 1 diabetes and nephropathy usually have other microvascular diseases, like retinopathy and neuropathy. Advanced retinopathy occurrance indicates histologic changes of DN in the glomeruli also .There is increased protein loss in urine of at least the microalbuminuric range.[37]

The relationship between diabetic nephropathy and retinopathy is less reliable in type 2 diabetes. One study with 35 patients of diabetes and significant proteinuria (>300 mg/d), 27 (77%) had diabetic nephropathy.[4] Retinopathy presented in 15 (56%) of the 27 with DN and in none of the 8 without DN. This gave retinopathy a sensitivity and specificity of 40% and 100% respectively.[5]

Type 2 diabetics with significant proteinuria and retinopathy are most likely have DN, but in those without retinopathy there is a higher incidence of nondiabetic glomerular disease .[5]

Diagnosis of diabetic nephropathy –

The techniques available for detecting diabetic nephropathy are the albumin : creatinine ratio or a protein:creatinine ratio from a random spot urine or a 24-hour urine collection[37] . Positive results need a second measurement to reconfirm. In diabetics protein excretion varies considerably. Urine dipsticks for microalbuminuria (on fresh morning specimens) are used only for initial screening. A positive tests should be followed-up by a 24-hour urine collection.[38] Microalbuminuria is defined as the urine protein : creatinine ratio of 30- 300 mg/g and macroalbuminuria is defined as a protein : creatinine ratio > 300 mg/g in a spot or 24 hours timed specimen . Short-term hyperglycemia, exercise, urinary tract infections, marked hypertension,heart failure, and acute febrile illness can cause transient elevations in urinary albumin excretion[38]. As there is a marked variability in the day to day excretion of albumin,at least 3 samples are required over a 3 month period ,before confirming the presence of microalbuminuria .[36]

Diagnosis of diabetic retinopathy

Early detection of diabetic retinopathy is essential .Photocoagulation therapy is more effective when applied to earlier stages of DR .Hence guidelines are required for the screening of patients ,so that retinopathy can be detected early.[38]

Current guidelines suggest that newly detected diabetics ,should undergo a fundus evaluation by an ophthalmologist soon after diagnosis of type 2 diabetes mellitus .The fundus examination should be done after full dilation of the pupils ,to visualise lesions even in the periphery of the retina .

Fundus findings

1. No retinopathy -

   No microvascular lesions.

2. Mild non-proliferative diabetic retinopathy (NPDR)

   Microaneurysms are main findings . 5% and 14% of patients progress to proliferative diabetic retinopathy in 1 year and 3 years respectively .

3. Moderate NPDR –

   Microaneurysms along with other microvascular lesions are seen.12–26% (within 1 year) and 30–48% (within 3 years) progress to proliferative diabetic retinopathy.[38]

4. Severe NPDR

Characterised by more than 20 intraretinal haemorrhages in four quadrants.Venous beading in two or more quadrants, or intraretinal microvascular abnormalities in one or more quadrant but not proliferative diabetic retinopathy. 52% (within 1 year) and 71% (within 3 years) progress to proliferative diabetic retinopathy.[39]

5. Proliferative diabetic retinopathy-

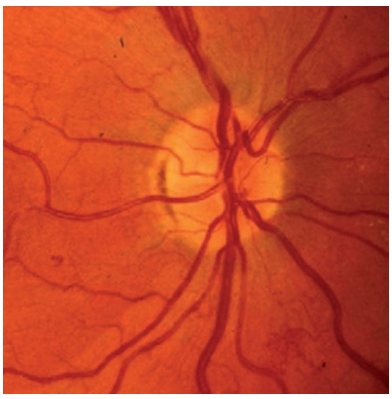
The findings noted are neovascularisation of optic disc (NVD) or elsewhere (NVE).Preretinal haemorrhages or vitreous haemorrhages.High risk characteristics are seen in those with vitreous haemorrhage irrespective of the degree of neovascularisation. Moderate NVE with vitreous haemorrhage is an indication for panretinal photocoagulation.[38]

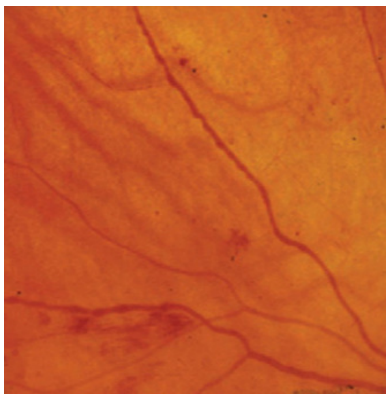
6. Clinically significant macular oedema -

Retinal thickening within 500 μm from centre of macula, hard exudates within 500 μm from centre of macula associated with adjacent retinal thickening. Retinal thickening of more than one optic disc area or within one optic disc diameter from centre of macula are considered characteristic of macular edema .[40]

(A) **(B)**

(E) **(F)** **(G)**





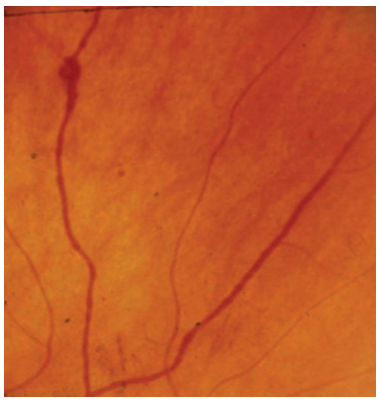


Fig A and B- denotes microaneurysms, haemorrhages and hard exudates .

Fig C – denotes intraretinal microvascular abnormalities(IRMA)

Fig D – denotes venous beading .

MATERIALS AND METHODS

Study type

Prospective study.

Sample size

157 cases

Inclusion criteria-

Patients with Type 2 Diabetes mellitus are included.

Exclusion criteria-

Type 1 Diabetics, known cases of nephrotic syndrome, active upper or lower urinary tract infections and chronic kidney disease.

Study Methods-

1. Diabetics were defined according to the American Diabetes association guidelines
    a. Fasting blood glucose >126 mg/dl, symptoms of hyperglycemia with a random blood glucose >200 mg/dl .
    b. Impaired fasting glucose defined as glucose between 100-126 mg/dl and a positive glucose tolerance test showing a 2 hours post prandial blood sugar >200 mg/dl are included.

2. Type 2 diabetics defined as patients on diet therapy or oral hypoglycemic agents for control of sugars. If on insulin therapy ,should have onset of diabetes after the age of 40 years ,with body weight excess than ideal at time of diagnosis.
3. Urine analysis and protein creatinine ratio of urine were obtained. Urine protein creatinine ratio to be confirmed on at least 3 urine samples.
4. If urinary tract infection is present ,treated according to urine culture report and then the proteinuria to be reassessed .
5. All diabetics are screened for retinopathy using direct opthalmoscopy by an ophthalmologist and graded according to the Early Treatment Diabetic retinopathy study. Fundus Fluorescein angiography (FFA) is to be done on those patients with evidence of diabetic retinopathy .
6. For all patients included in the study,serum creatinine estimation to be done, and the estimated GFR calculated using modified MDRD equation .
7. Renal biopsy was done in those patients who had,
    i. Absence of diabetic retinopathy .
    ii. Rapid decline of GFR .
    iii. Nephrotic range proteinuria with a normal GFR .
    iv. Active urinary sediments .

Renal biopsy was done after obtaining an informed written consent.

Biopsies were interpreted by a single pathologist under light microscopy using hematoxylin and eosin and periodic acid Schiff(PAS) staining .

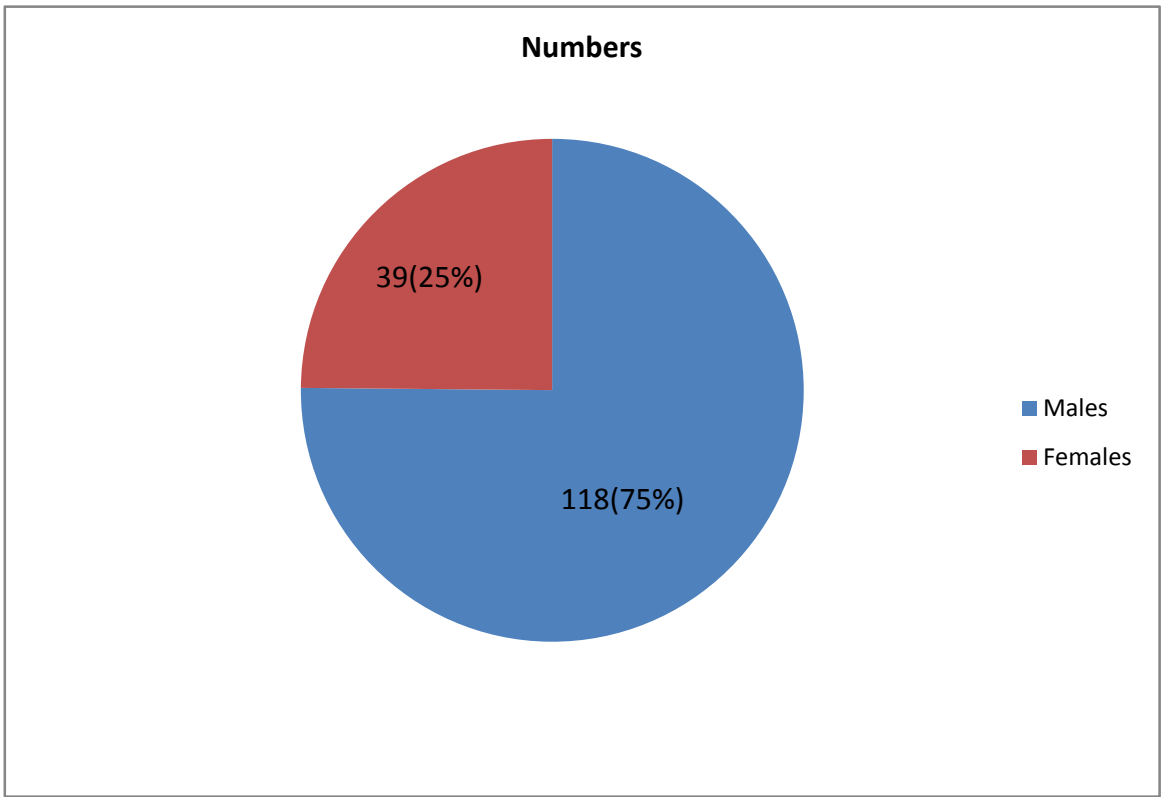
RESULTS

Total number of Type 2 diabetics studied – 157

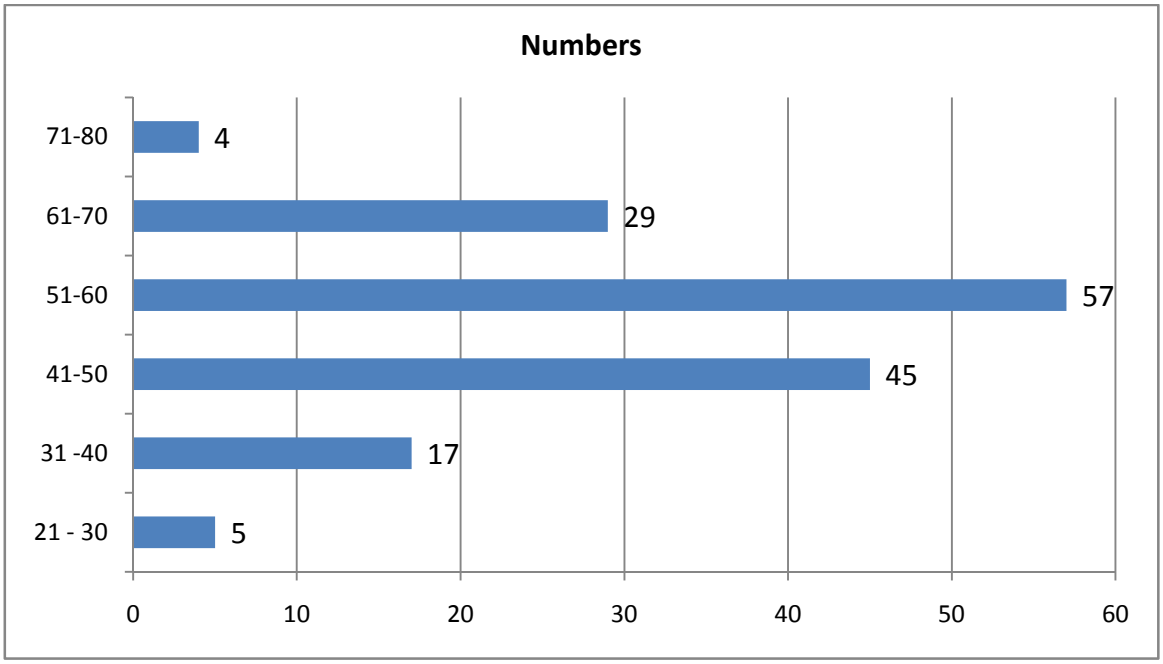
Sex distribution -

Males comprised of 118 patients (75%) of the study group.

Females comprised of 39 patients(25%)



Age distribution

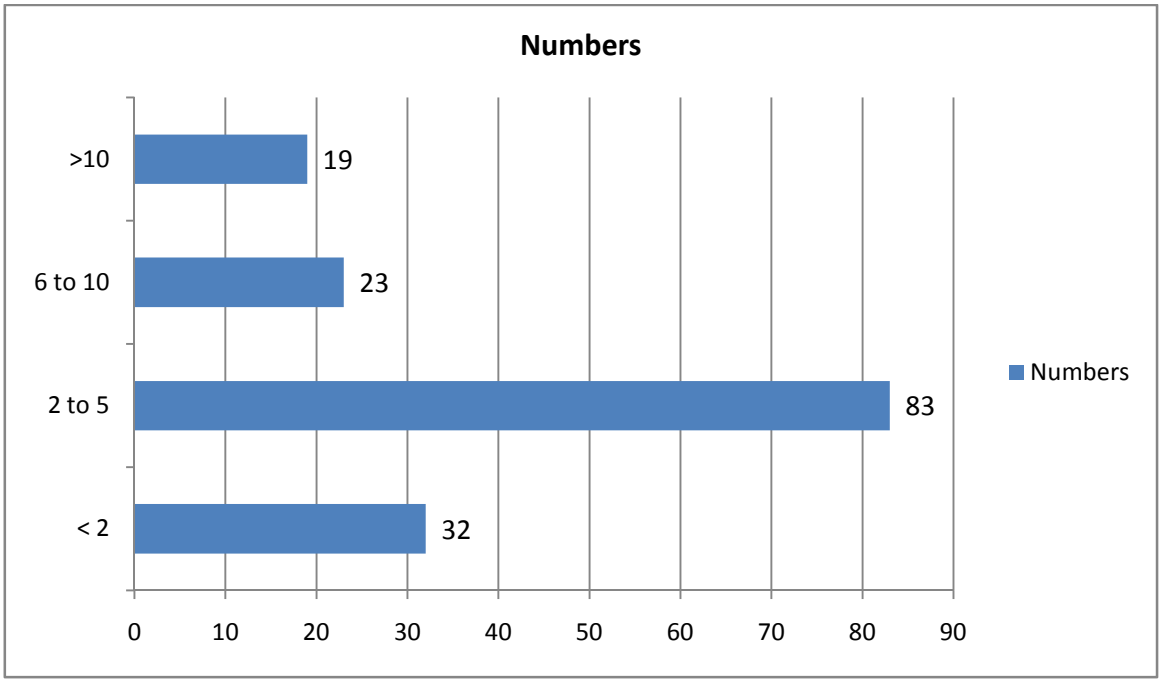


Age distribution frequency –

Age range(years)	Numbers	Percentage %
21-30	5	3
31-40	17	10.8
41-50	45	28.7
51-60	57	36
61-70	29	18.5
71-80	4	2.5

The age in the study population ranged from 26 to 78 years ,with a mean age of 52 years with a SD of 10 years .

Duration of diabetes –



In a majority of patients duration of diabetes was between 2 to 5 years comprising 53 %.

Duration was less than 2 years in 20 % and more than 10 years in 12% .

The least duration of diabetes in the study group was 2 months ,and the maximum duration was of 25 years .

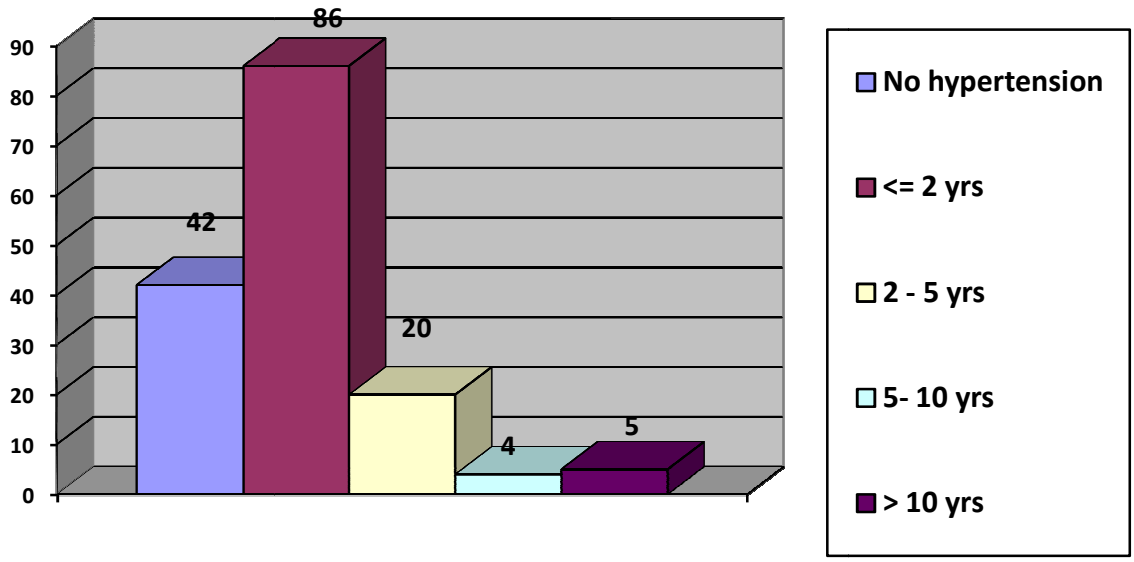
The mean duration of diabetes was 5.59 years with a SD ±5.1 .

Hypertensive profile of the study group –

Total of number of patients with hypertension = 115(73%)

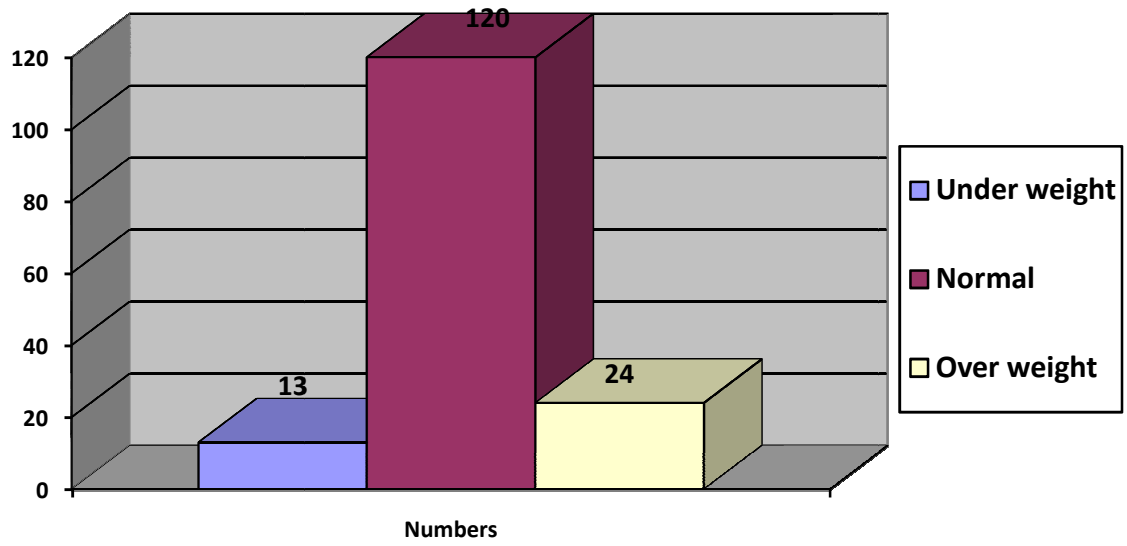
Total number without hypertension =42(27%)

Profile of Hypertension



The mean duration of hypertension was 1.9 years with a standard deviation of 2.9 years .

BMI of the study population –



13(8%) were underweight with a BMI less than 18 .

24(15%) were overweight with a BMI of more than 25 .

The BMI ranged from 16 to 30 ,with a mean BMI of 22.1 and a SD ±2.4

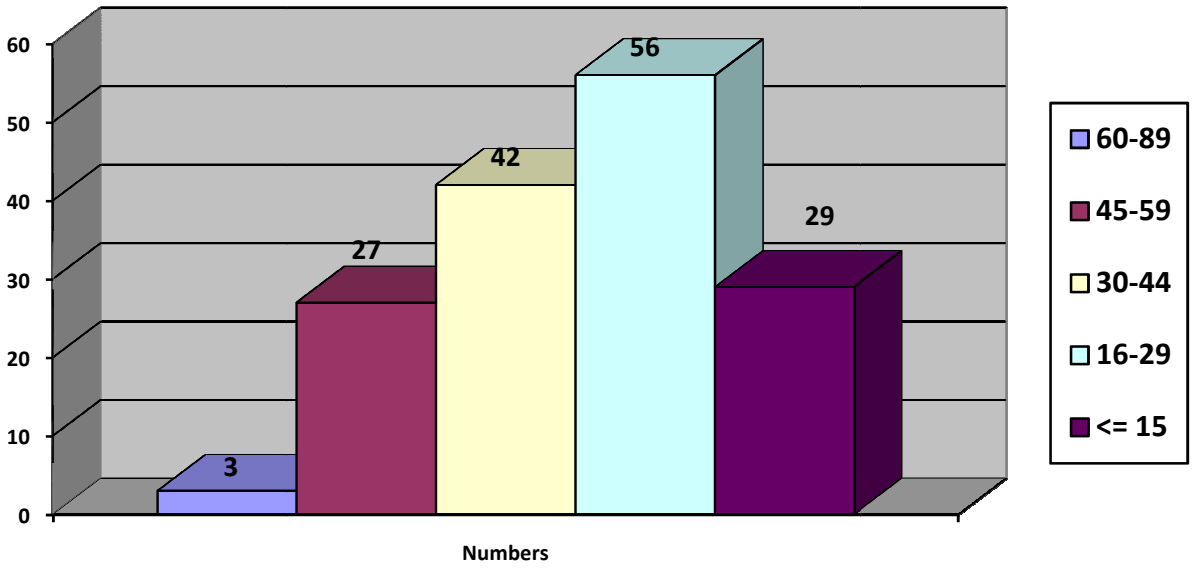
GFR profile –

For all patients in the study group GFR was measured using modified MDRD equation .

The GFR ranges from 5 ml/min to 86 ml/min .

Mean GFR is 29 ml/min with a SD ±15 .

GFR profile and frequency curve -



Profile of diabetic microvascular complications –

- Nephropathy (proteinuria)
- Retinopathy
- Neuropathy

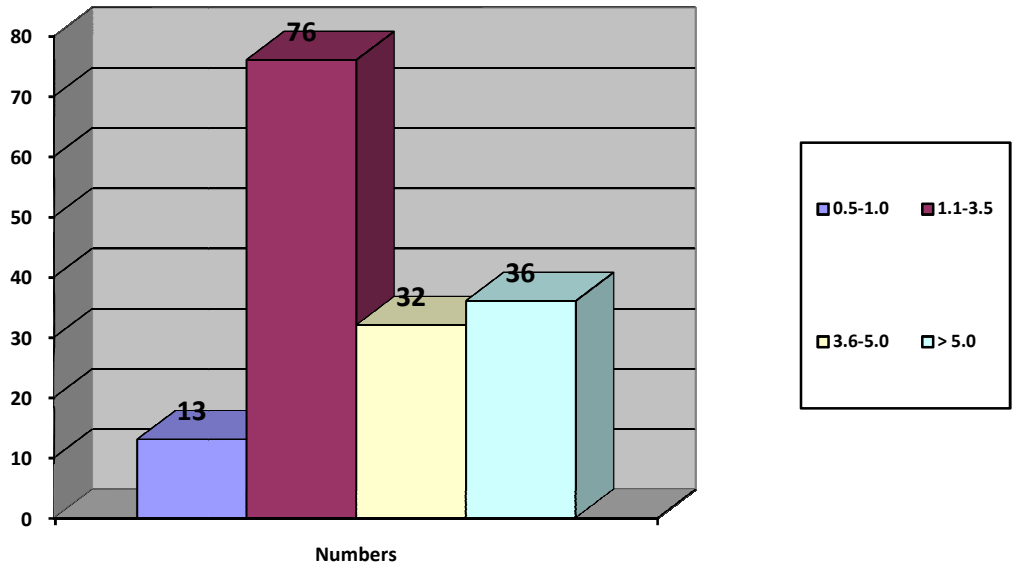
Profile of Proteinuria –

All the diabetics in the study population had proteinuria .

Urine protein creatinine ratio (PCR) was done .

The PCR ranged from 0.3 g/mg - 8.1 g/mg ,with a mean PCR of 3.6 g/mg and a standard deviation of 2.0 g/mg .

PCR FREQUENCY CURVE-



Urine analysis showed an active sediment with RBC or RBC casts in 6 out of the 157 patients.

Profile of Retinopathy –

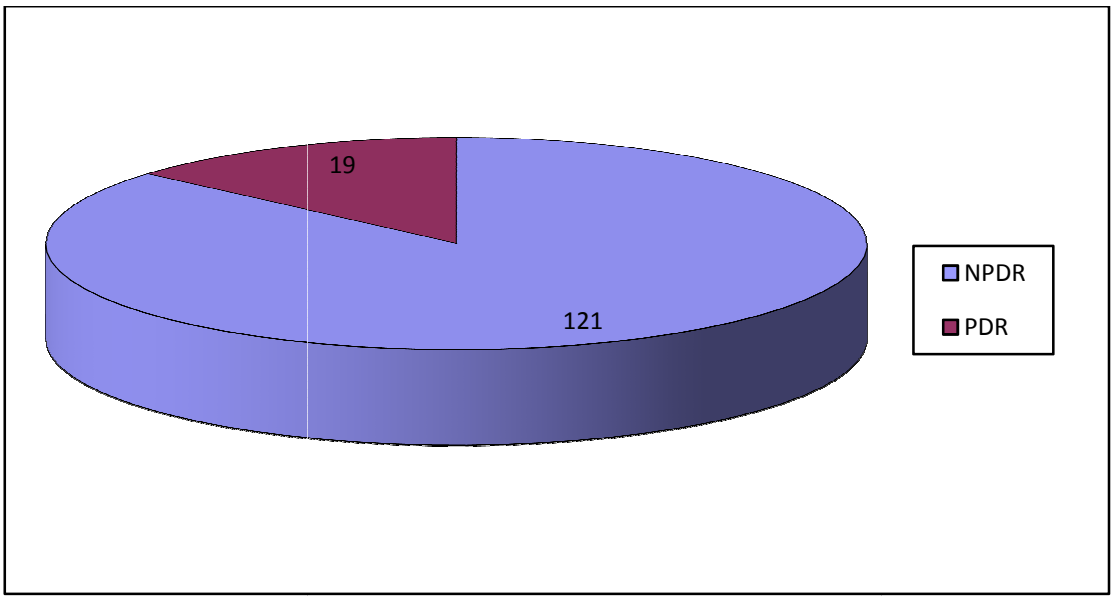
140 patients in the study group had evidence of diabetic retinopathy .

Non proliferative diabetic retinopathy(NPDR) was seen in 121

Proliferative diabetic retinopathy(PDR) was seen in 19 .

In 94 of the 140 patients ,retinopathy was confirmed by fundus fluorescein angiography (FFA)

20 patients underwent panretinal photocoagulation (PRPP).



ASSOCIATION BETWEEN RETINOPATHY AND PROTEINURIA-

The association between retinopathy and the quantum of proteinuria using urine PCR was analysed .

Retinopathy	Urine Protein Creatinine Ratio (PCR) mg/g			
	0.5 - 1	**1.1-3.5**	**3.6-5**	**>5**
Yes	9.3 %	47.9%	17.9%	25%
No	0	52.9%	41 %	6%

p=0.04

Analysis showed 25 % of patients with diabetic retinopathy had a quantum of proteinuria (PCR) > 5 mg/g ,whereas in the non diabetic retinopathy group only 6 % had this quantum of proteinuria.

This difference was statistically significant (p value =0.04)

ASSOCIATION BETWEEN DIABETIC RETINOPATHY AND HYPERTENSION –

We analysed the study population to see if retinopathy was more common in t patient having co existing hypertension as well .

	Hypertension	
Retinopathy	**Yes**	**No**
Yes	103 (73%)	37(26 %)
No	10 (58%)	7 (42 %)

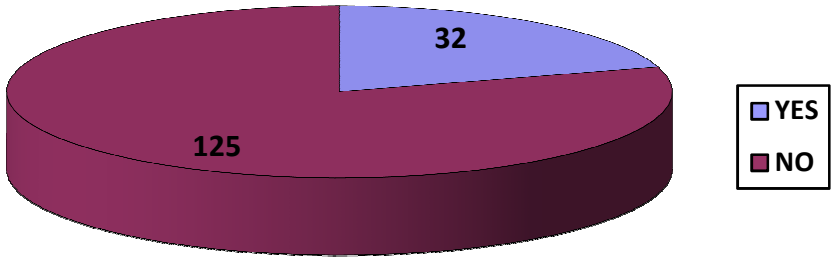
p =0.2

73 % of the patients with retinopathy, had coexisting hypertension also compared to 26% with retinopathy who did not have hypertension .

This difference however was not statistically significant (p=0.2)

Profile of neuropathy –

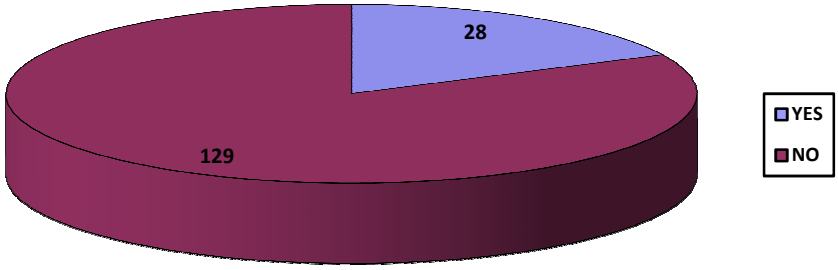
Diabetic peripheral neuropathy was seen in 32 patients (20 %) of the study population .



Profile of macrovascular diabetic complication –

Ischaemic heart disease

Ischaemic heart disease in the form of left ventricular dysfunction was seen in 28 patients (17%) of the study group .



Profile of renal biopsy-

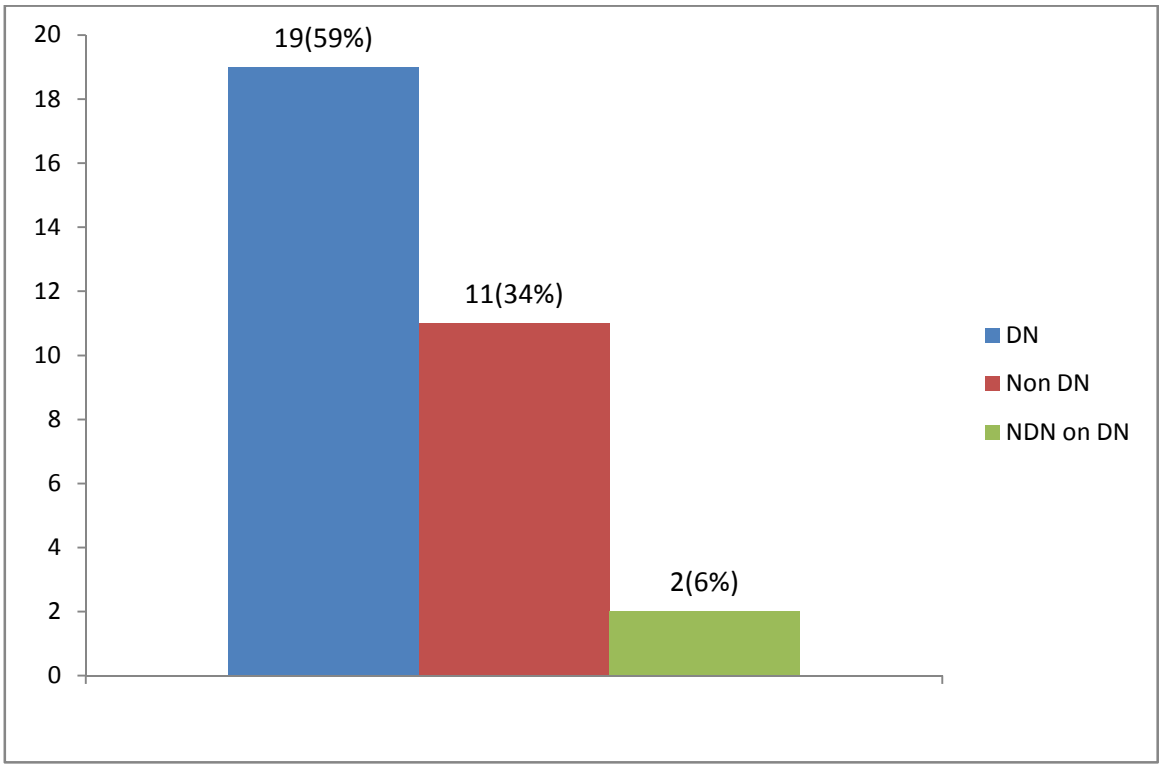
Renal biopsy was done in 32 patients(20.4%) of the study population based upon the indications for renal biopsy as mentioned in the study protocol .

The biopsies (n=32) showed

Diabetic nephropathy(DN) - 19 (59 %)

Non diabetic nephropathy (Non DN) - 11 (34%)

Non diabetic nephropathy superimposed on DN - 2 (6 %)



Spectrum of non diabetic nephropathy –

Pathology	Frequency
Membranous Nephropathy	2
MCD/FSGS	2
Crescenteric glomerulonephritis	1
Anti –GBM disease	1
Acute tubular necrosis (ATN)	1
Chronic interstitial nephritis (CIN)	1
Glomerulosclerosis	2

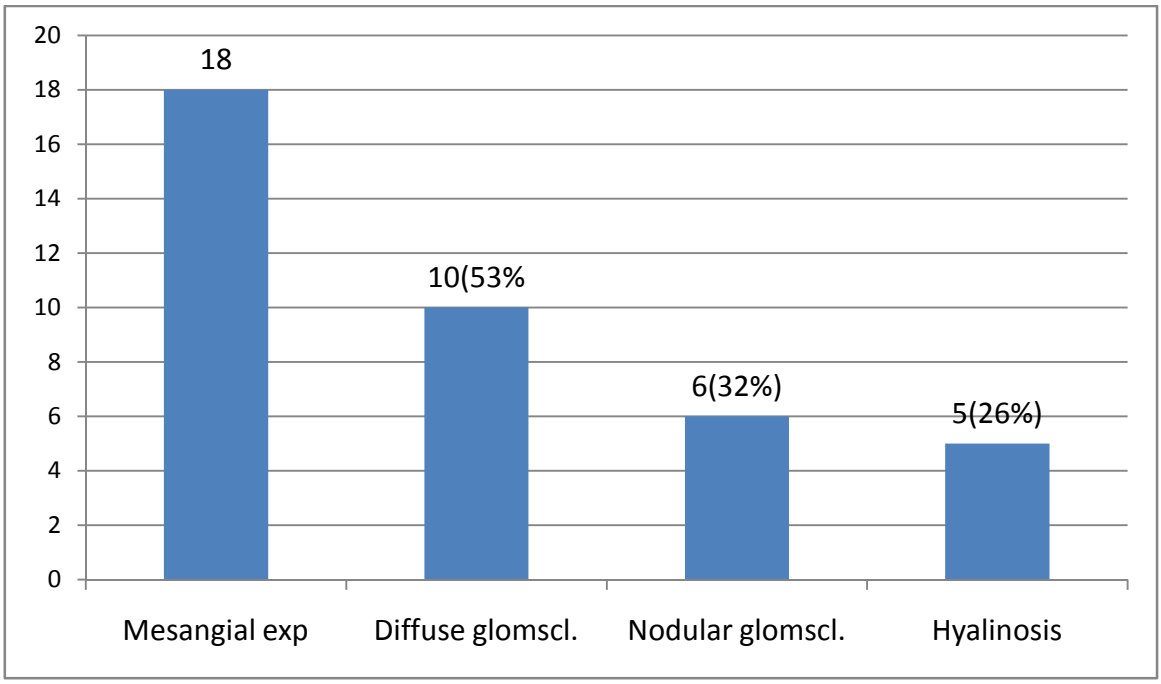
The patterns of non diabetic kidney disease superimposed on diabetic nephropathy (n=2) were

- Infection related glomerulonephritis (IRGN) with diabetic nephropathy .
- Henoch Scholein purpura (HSP) with diabetic nephropathy .

Pathological features of diabetic nephropathy –

The pathological features of diabetic nephropathy noted on renal biopsy were ,

Pathology	Frequency(n=19)
Mesangial expansion	18
Diffuse glomerulosclerosis	10(53%)
Nodular glomerulosclerosis	6(32%)
Hyalinosis of Efferent artery	5(26%)



Analysis of risk factors for diabetic nephropathy–

The risk factors for the cases of biopsy proven diabetic nephropathy were analysed.

Risk factors analysed are -

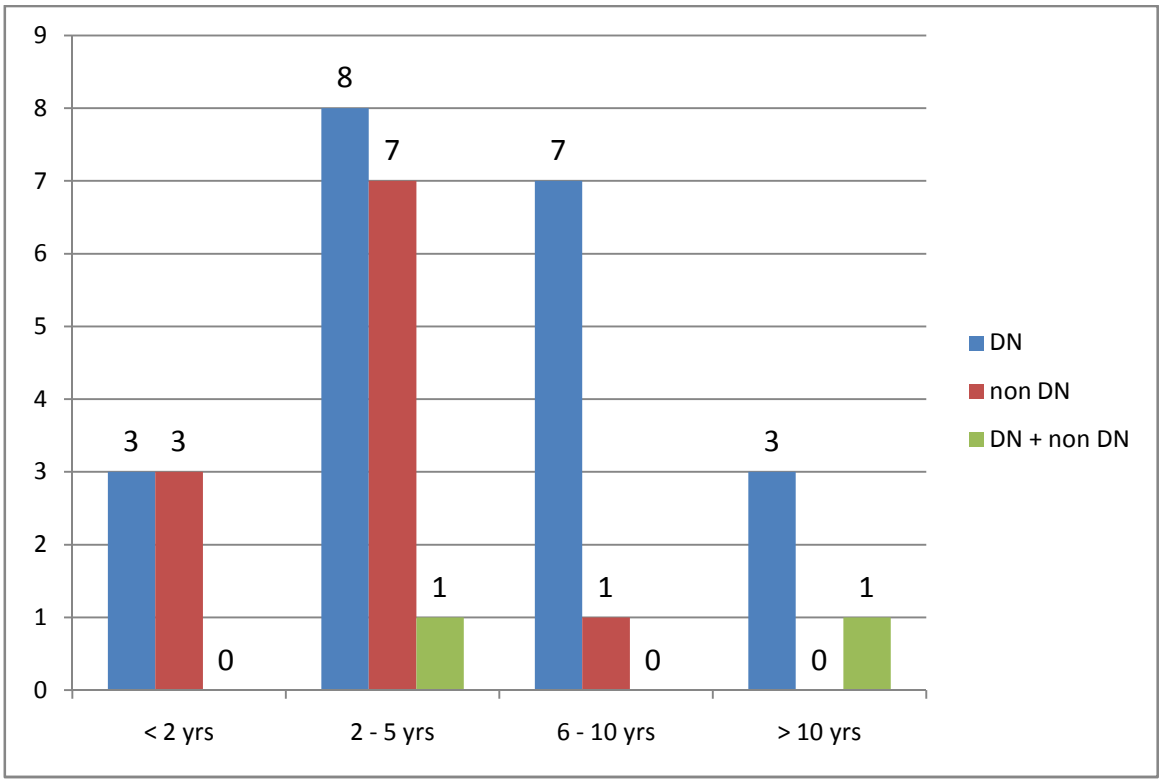
1. Duration of diabetes.
2. Patient age
3. Quantum of proteinuria
4. Hypertension
5. BMI
6. Esimated GFR(eGFR)
7. Serum cholesterol

Relation between duration of diabetes and diabetic nephropathy -

The prevalence of diabetic nephropathy based on the duration of diabetes was analysed .The duration of diabetes in the diabetic nephropathy and non diabetic nephropathy group was as follows .

Duration (yrs)	**DN (n=19)**	**Non DN (n=11)**	**DN + nonDN (n=2)**
< 2	3(15%)	3(27 %)	0
2- 5	8(42%)	7(63%)	1
6- 10	7(36%)	1 (9%)	0
>10	3(5%)	0	1

Graphical representation of duration of diabetes in diabetic nephropathy Vs non diabetic nephropathy .



The statistical analysis of duration of diabetes as a risk factor for diabetic nephropathy was done by a Chi Squared test .The analysis showed that duration of diabetes was not a statistically significant risk factor (p value 0.8).

Thus in our study, duration of diabetes was not a predictor of diabetic nephropathy .

2. Relation between patient age and nephropathy –

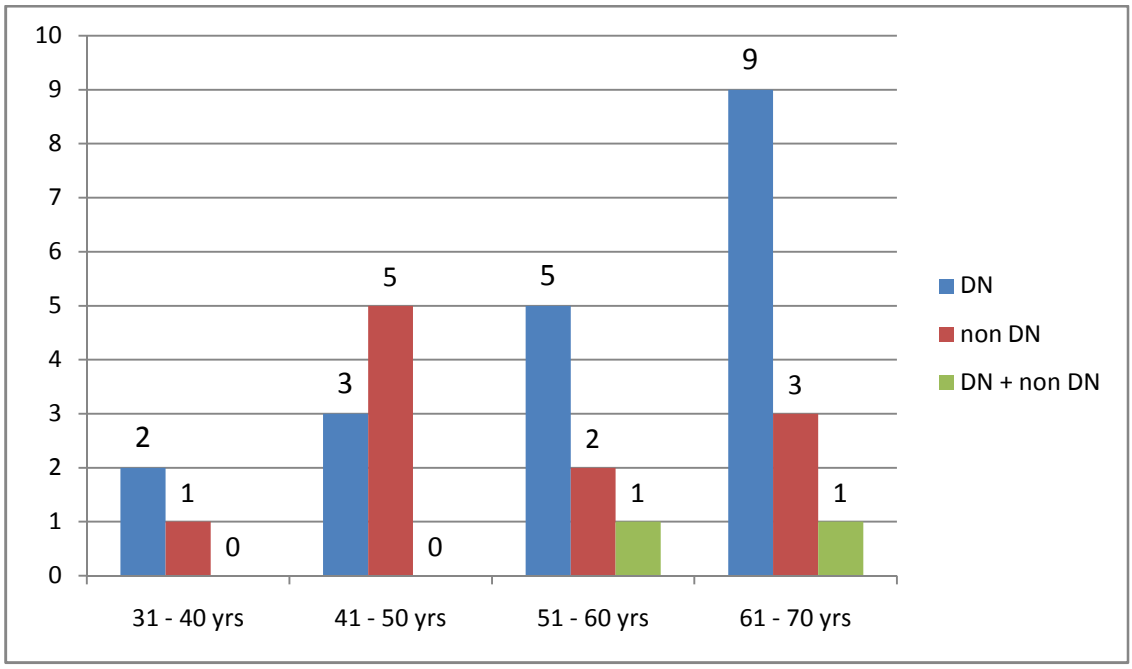
The age group of 61 – 70 years comprised the largest group of patients with diabetic nephropathy(n=19) in the study ,8 patients (42 %) were in this group,followed by the age group of 51 – 60 years comprising 5 patients (26 %) .The age groups (31 – 40) and (41 – 50) comprised of 10 % and 15 % of the study population respectively .

Relation between patient age and non diabetic nephropathy –

In the non diabetic nephropathy group(n=11) ,the age group 41 – 50 years was the most common, comprising 5 patients (45.5%).The age groups 51 – 60 yrs and 61 – 70 years comprised 18 % each respectively.

Comparision of patient age as a risk factor in the DN vs non DN groups.

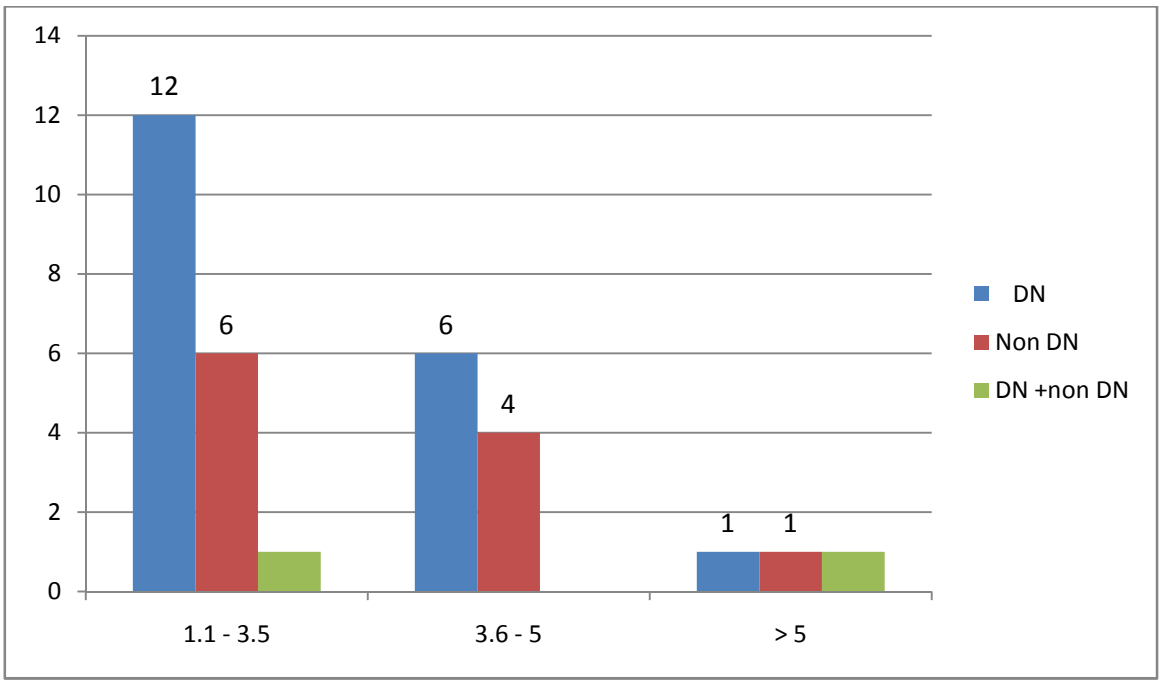
Analysis using the chi squared test ,showed that age is not a statistically significant risk factor for the development of diabetic nephropathy **(p =0.7)**



Relation between proteinuria and diabetic nephropathy –

Urine protein estimation was done using the spot urine protein creatinine ratio (urine PCR) .The quantum of proteinuria and its relation with diabetic nephropathy was analysed .

Urine PCR (g/mg)	DN (n=19)	Non DN (n=11)	DN + non DN (n=2)
1.1 – 3.5	12 (63%)	6 (55%)	1
3.6 – 5	6 (31%)	4 (36%)	0
> 5	1 (5%)	1(5%)	1



Comparison of quantum of proteinuria in diabetic nephropathy vs non diabetic nephropathy –

The quantum of proteinuria was compared in the diabetic nephropathy vs non diabetic nephropathy group .

Statistical analysis using the Chi Squared test ,showed *the p value* to be **0.3** which was not statistically significant.

We were unable to differentiate diabetic nephropathy from non diabetic nephropathy based on the quantum of proteinuria in our study .

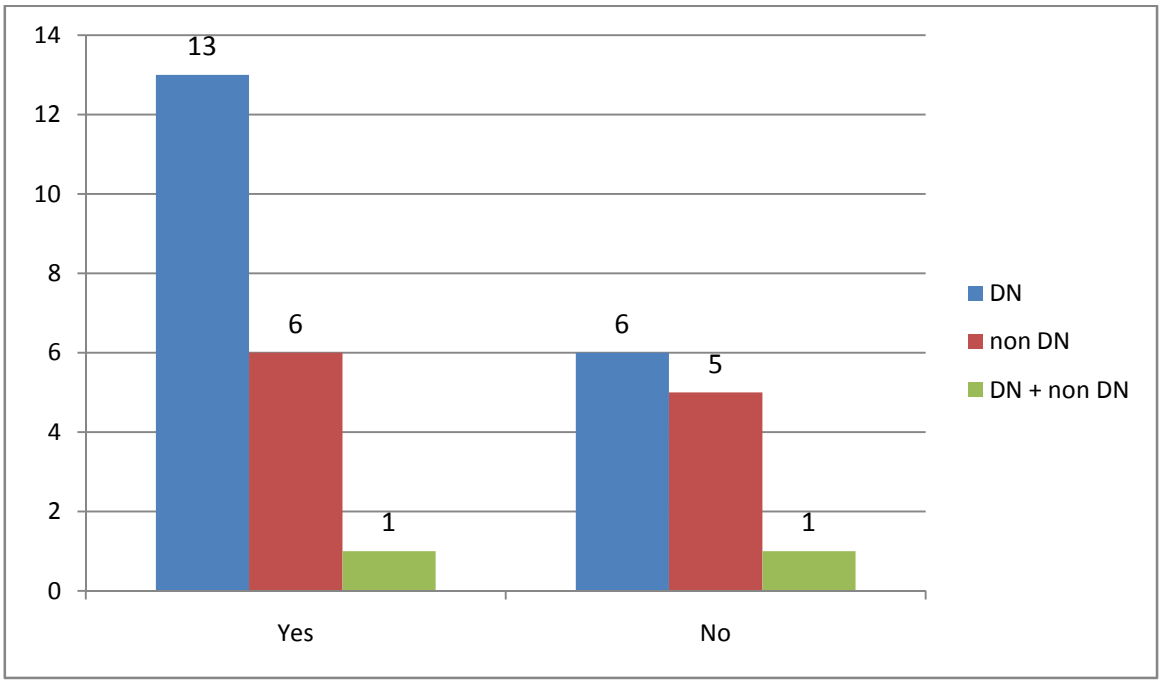
Relation between Hypertension and diabetic nephropathy –

The presence and duration of hypertension were analysed as risk factors for diabetic nephropathy . The prevalence and duration of hypertension in the diabetic vs non diabetic nephropathy group were analysed.

The prevalence of hypertension was as follows

Hypertension	DN (n=19)	Non DN (n=11)	DN + non DN
Yes	13 (68 %)	6(55 %)	1
No	6(32 %)	5(45%)	1

Hypertension profile-

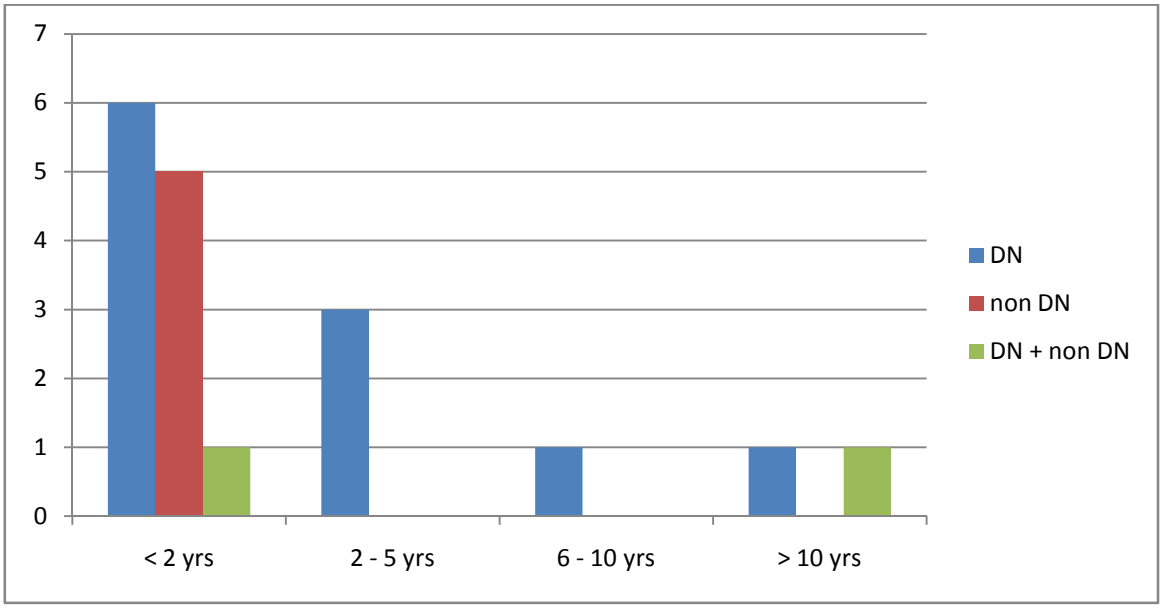


Analysis of duration of hypertension –

The analysis of duration of hypertension showed the following data

HTN duration	DN(n=19)	Non DN(n=11)	DN + non DN (n=2)
< 2 yrs	6(31 %)	5(45 %)	1
2 – 5 yrs	3(16%)	0	0
6 – 10 yrs	1(5%)	0	0
>10 yrs	1(5%)	0	1

Graphical representation-



Analysis of hypertension as a risk factor for diabetic nephropathy –

The statistical analysis of hypertension as a risk factor for diabetic nephropathy was done using the Chi Squared test.

The result showed that hypertension and its duration were not statistically significant risk factors with *a p value* of **0.7 and 0.19** respectively **.**

Analysis of BMI as a risk factor for nephropathy –

The BMI profile for the patients in the DN vs non DN group was follows .

BMI	**DN**	**Non DN**
Underweight(BMI<18)	1(5.3%)	1(9 %)
Normal (19 -24)	18 (94.7 %)	10 (91%)
Overweight(>25)	0	0

p value=0.85

There was no significant difference in BMI between the DN and non DN group in the study (p value 0.85).

Estimated GFR(eGFR) comparision between DN vs non DN group –

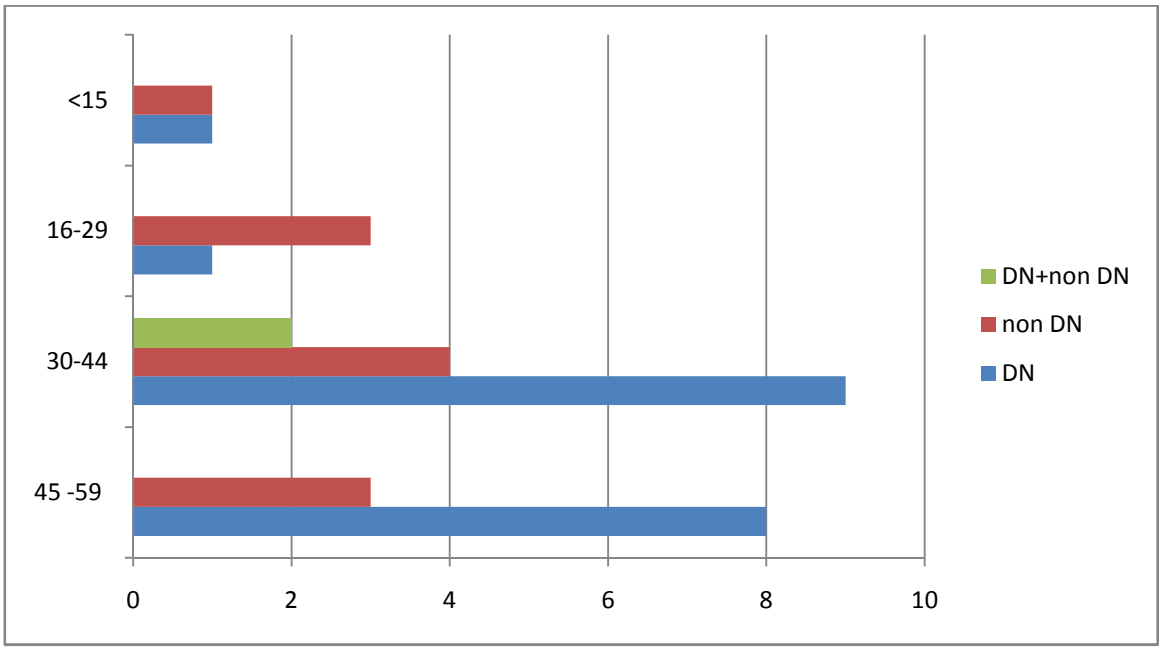
GFR estimation was done in diabetic nephropathy (DN) and non diabetic nephropathy (NDN) group using the modified MDRD equation.

The following results were noted

eGFR(ml/mt)	DN(n=19)	Non DN(n=11)	DN +non DN(n=2
45 - 59	8(42%)	3 (27%)	0
30 - 44	9(47%)	4(36%)	2(13%)
16-29	1(5%)	3(27%)	0
<15	1(5%)	1(9%)	0

p=0.4

There was no statistical difference in eGFR between the DN and non DN group (p value 0.4).



Comparision of cholesterol profile in the DN Vs Non DN group –

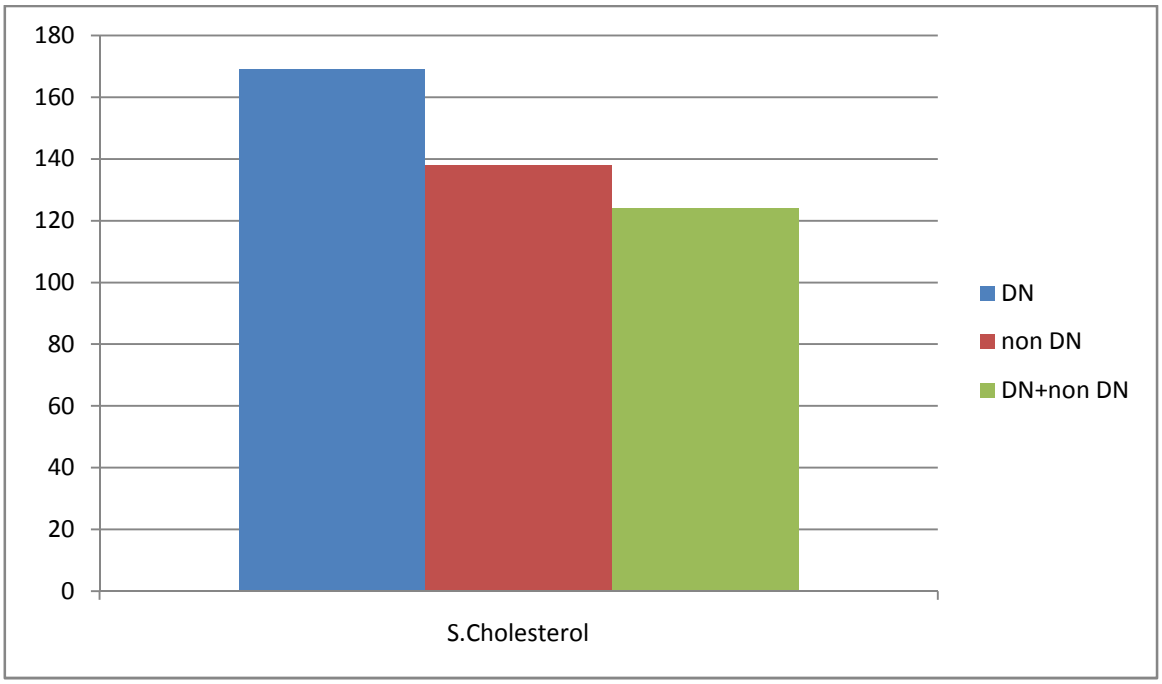
The serum cholesterol levels in diabetic nephropathy and non diabetic nephropathy group were as follows .

Cholesterol profile-

Group	Mean cholesterol(mg/dl)	Range(mg/dl)
DN	160	110-234
Non DN	138	118-186
DN + non DN	123	120-126

p = 0.19

Though the mean serum cholesterol levels were higher in DN group compared to the non DN group ,this difference was not statistically significant (p=0.19)



Concordance of diabetic retinopathy and diabetic nephropathy –

The presence of diabetic retinopathy was evaluated in the diabetic nephropathy(DN), non diabetic nephropathy(NDN) and the NDN superimposed on DN group .

The results in each group were as follows .

Diabetic retinopathy	**DN (n=19)**	**NDN (n=11)**	**DN + NDN (n=2)**
Yes	10(53%)	4(36%)	1
No	9(47%)	7(63%)	1

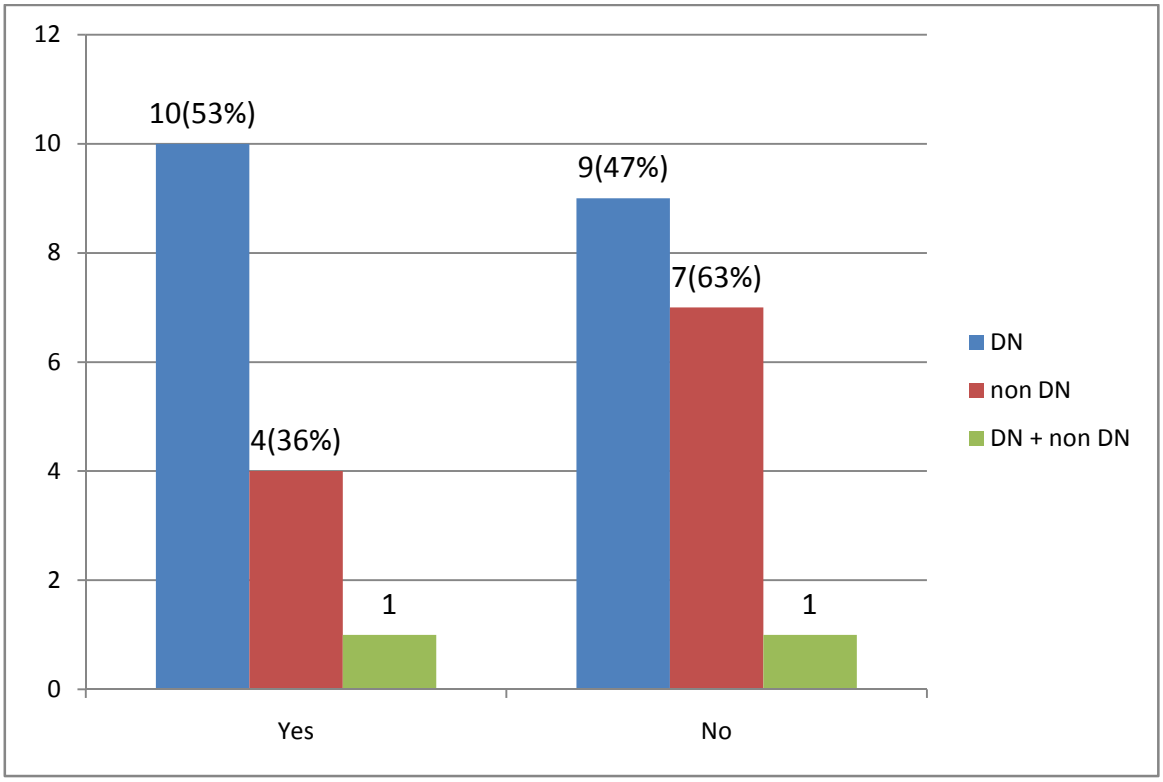
Total num. of patients with biopsy proven diabetic nephropathy - 21

Total num .with retinopathy in this group - 11

Percentage with diabetic nephropathy having retinopathy - 54%

Percentage without diabetic nephropathy having retinopathy - 36 %

Graphical representation of diabetic retinopathy in DN vs NDN groups-



Statistical analysis by Chi Squared testing showed that even though retinopathy was seen in 53 % of patients with diabetic nephropathy and in only 36 % of patients with non diabetic nephropathy ,this difference was **not** statistically significant (p value **0.6**).

Sensitivity of retinopathy for diabetic nephropathy is 71.4%

Specificity of retinopathy is 63.6 %

DISCUSSION

Type 2 diabetes is a global epidemic with an increasing incidence over the last decade. Diabetic nephropathy (DN) occurs in approximately one third of the patients with type 2 diabetes and contributes greatly to its morbidity ,mortality and cost of treatment.

It is diagnosed clinically by persistant proteinuria (>500 mg/day), hypertension and the presence of diabetic retinopathy in a patient with diabetes of long standing duration.

In addition to classical diabetic nephropathy ,diabetic patients can also develop primary or secondary glomerular diseases unrelated to diabetes (NDN). There exists a third group of patients also who can have a NDN superimposed on a background of DN.

We suspect a NDN when a diabetic presents with overt proteinuria with duration of diabetes being less than 5 years, a rapid decline in renal function, impaired renal function in the absence of significant proteinuria,active urinary sediment or the absence of diabetic retinopathy.

The presence of diabetic retinopathy while correlating well with diabetic nephropathy in type 1 diabetics ,the concordance in type 2 diabetics is only around 60 % as demonstrated in the study by Parving[4] et al published in 1992.Study by Prakash[3] et al published in 2007 showed

retinopathy to be a poor predictor of diabetic nephropathy as 50 % of the renal biopsy proven diabetic nephropathy patients in their study lacked retinopathy.

This background led us to our present study ,were we studied the concordance between diabetic nephropathy and retinopathy, the clinical and lab profile of a patient with diabetic nephropathy Vs a patient with non diabetic nephropathy and the spectrum of non diabetic renal diasease in a proteinuric type 2 diabetic .

In our study we analysed 157 proteinuric type 2 diabetics .The study population comprised of 118 males (75 %) and 39 females (25 %) ,with a male to female ratio of 3:1. The male predominance in proteinuric diabetic patients has also been documented in the study by Parving[4] et al which showed a male to female ratio of 4:1 .

The age group of the study population ranged from 26 years to 78 years with a mean age of 52 years .Majority of the patients were in the age group of 51 – 60 years comprising of 57 patients (36 %) followed by the age group 41 to 50 years which had 45 patients (28 %) . The study by J Prakash[3] et al analysing diabetic retinopathy as a predictor of nephropathy ,showed an age range of 30 – 78 years with a mean age of 54 years, comparable to our study.

Analysis of the duration of diabetes showed ,the duration to range from a minimum of 2 months to a maximum duration of 25 years .The mean duration was 5.59 years with a standard deviation of 5.1 years .In a majority the duration of diabetes was between 2 to 5 years comprising 82 patients(53 %).The Parving study has not evaluated the duration of diabetes while the J Prakaash study showed a mean duration of diabetes of 10 years with a range from 4 months to 25 years .The mean duration of diabetes was substantially lower in our study.

Another study by Teresa Wong[40] et al showing the profile of proteinuric diabetics with and without diabetic nephropathy showed a mean duration of diabetes of 8 years in their study.

In our study group 115 patients (73 %) had coexisting hypertension as well.The mean duration of hypertension was 1.9 years +/- 2.9 years . In a majority 86 patients (56 %) the duration of hypertension was less than 2 years .J Prakash et al study showed a hypertension prevalence of 47.8% in their study group .In the Teresa[40] et al study the prevalence of hypertension was 87 % which was comparable with our study . Dakshinamoorthy[41] et al study of non diabetic kidney disease in a proteinuric type 2 diabetic, showed a prevalence of hypertension of 84 % in their study.

The nutrition status of our study population was assessed by body mass index measurements (BMI) .The mean BMI was 22.1 (range from 16 to 30).13 patients (8 %) had a BMI less than 18 and were classified as underweight .24 patients had a BMI of more than 25 and were classified as overweight .Teresa[40] et al showed a mean BMI of 24.9 in their study group.

For all our patients we estimated the glomerular filtration rate (GFR) using the modified MDRD equation .The mean GFR was 29 ml/min with a standard deviation of 15 ml/min. GFR range was from 5 – 86 ml/min .In the Teresa[40] et al study the mean serum creatinine values were 167 ± 100 µmoles, GFR estimations were not done. The mean serum creatinine in the Dakshinamoorthy[41] et al study was 4.3 mg/dl ± 3.9 mg/dl.

The quantum of proteinuria in the study group was determined by urine protein creatinine ratio (PCR).The urine PCR ranged from 0.3 g/mg to 8.1 g/mg ,with mean PCR of 3.6 g/mg .The mean quantum of proteinuria in the Dakshinamoorthy study was 4.2 gram/day and was 2 gram/day in the Theresa et al study.

The presence of diabetic retinopathy was determined for all patients in the study population. 140 patients(89 %) had diabetic retinopathy . Non proliferative diabetic retinopathy was seen in 121 patients (86 %) .Proliferative diabetic retinopathy was seen in 19 patients (14 %).In 94 of

the 140 patients retinopathy was confirmed by fundus fluorescein angiography(FFA).

20 patients underwent pan retinal photocoagulation (PRPP) for proliferative diabetic retinopathy .In a study by Trevisan[47] et al in 2002 evaluating the effect of retinopathy and concomitant proteinuria in a diabetic patient, showed 55 % of the proteinuric(>500 mg/day) diabetics to have retinopathy .In their cohort 52 % had proliferative diabetic retinopathy and 48 % non proliferative diabetic retinopathy .We analysed the association between retinopathy and quantum of proteinuria .We found that 25 % of patients with retinopathy had a urine PCR>5 g/mg while only 6 % without retinopathy had this quantum of proteinuria .This difference in PCR between the two groups was found to be statistically significant .Trevisan[47] study also found a difference in proteinuria (1.9 g/day vs 0.8 g/day) between the retinopathy and non retinopathy group though not statistically significant.

Hypertension as a risk factor for retinopathy was analysed in our study,showed that 73 % patients with retinopathy had coexisting hypertension also, but this relation was found not be statistically significant. Trevisan et al also found this association not to be statistically significant.

Evaluation for other microvascular and macrovascular complications of diabetes showed presence of peripheral neuropathy in 32 patients (20 %) and ishaemic heart disease presenting as LV dysfunction in 28 patients (17 %).

Renal biopsies were done in 32 patient (20 %) of the study population based on the biopsy protocol of our study .The biopsy results showed diabetic nephropathy(DN) to be present in 19 patients (59 %) of the total number of 32.

Non diabetic nephropathy (NDN) was seen in 11(34%) patients and non diabetic nephropathy superimposed on a background of diabetic nephropathy(NDN +DN) was seen in 2 patients(6%).

The non diabetic nephropathies (NDN=11) seen in the study group were membranous nephropathy (2),focal segmental glomerulosclerosis (FSGS) in (2), cresenteric glomerlonephritis(1) and anti GBM disease(1) , tubulointerstitial nephritis ATIN (1) and CTIN(2) and finally global glomerulosclerosis (with no evidence of DN) suggestive of chronic kidney disease in (2).

The patterns of non diabetic nephropathies(NDN) superimposed on DN, (NDN+DN =2) were Infection related glomerulonephritis(IRGN) with DN and Henoch Scholein purpura with DN.

The pathological features of diabetic nephropathy observed were as follows ,mesangial expansion in 18,diffuse glomerulosclerosis in 10 ,nodular glomerularsclerosis in 6 and efferent arteriolar hyalinosis in 5.

The prevalence of non diabetic kidney disease in a proteinuric diabetic varies widely. Most Indian studies showed a prevalence from 30 % to 50 % .This wide variation in the incidence of non diabetic kidney disease, can be due to the differences in biopsy protocols for proteinuric diabetics in different units .The Dakshinamoorthy[41] study showed a very high prevalence of NDN of 64 % .The biopsy protocol they followed was similar to ours but they had a significantly longer study period of 18 years and theirs was a retrospective study .Study by GT John[42] et al in 1994 ,showed the prevalence of NDN to be 50 % in their study.

In Prakash[3] et al study the prevalence of NDN was 43 % .Their study showed 50% of the NDN patients to have glomerular disease and the remaining 50 % tubulointerstitial disease .Membranous nephropathy was the most common glomerular disease in their study .The GT John[42] study from south India showed proliferative glomerularnephritis to be the commonest NDN .Premlatha[44] study showed showed membranous nephropathy to be the most common NDN ,in their study .These results show that the prevalence of different primary and secondary glomerular diseases(NDN) in

a diabetic, depends on the usual prevalence of these diseases based on their geographical and ethinic distribution , hence is a coincidental finding. As per Bertani T[46] et al the predisposition of DN to superimposed glomerularnephritis is due to the increased exposure to antigenic components causing immune response .Regarding the renal lesions in diabetic nephropathy ,the most common lesion was found to be diffuse glomerulosclerosis followed by nodular glomerulosclerosis in around 30 %. Prakash[3] et al showed diffuse glomerosclerosis in 62 % and nodular sclerosis in 37 % respectively.

Risk factors in the biopsy proven diabetic nephropathy group were analysed ,they were 1)duration of diabetes, 2)patient age, 3)quantum of proteinuria, 4)presence of hypertension, 5)body mass index(BMI), 6)estimated GFR and 7)serum cholesterol levels .

1) Duration of diabetes

In both the DN and non DN group the most common duration of diabetes was between 2 to 5 years ,comprising 42 % and 63 % respectively. The duration of diabetes on analysis was found not to be significant risk factor for DN in our study. Teresa et al study showed a longer duration of diabetes in the group having DN compared to the non DN ,and this was significant in her study .However in other studies by Gwie[45] et al and Soni[43] et al showed no significance of duration of diabtetes on presence of DN .

2) Patient age

The age group of 61 to 70 years was the most common age among patients of the DN group (42%) followed by the age group 51 to 60 years .In the non DN group the age group 41 to 50 years was the most common. Analysis of age as a risk factor for DN was found to be not statistically significant in our study .This finding was comparable to the Teresa[40] ,Gwie[45] and Soni[43] studies also which showed no statistical significance of patient age on DN .

3) Quantum of proteinuria

The quantum of proteinuria was similar (PCR 1.1 to 3.5 g/mg) in both the DN and non DN group comprising 63 % and 55 % respectively.The quantum of proteinuria was found not to be a significant factor to differentiate DN from non DN .This observation was made by both the Teresa[40] et al and Dakshinamoorthy[41] study which showed no significance of quantum of proteinuria on predicting DN .

4) Hypertension

The presence of hypertension was analysed in both the DN and the non DN group. It was seen in 68 % and 55 % of patients in the DN and non DN group respectively which was not statistically significant .Teresa study was also of the similar opinion ,however the Dakshinamoorthy study

demonstrated a significant increase in the prevalence of hypertension in the DN group compared to the non DN group .

5)Estimated GFR(eGFR)

45 % of the DN group had an eGFR of (45 to 59 ml/mt) while in the non DN group only 27 % were in this range of eGFR .We also found that 27 % in the non DN group had an eGFR in the range of (16 – 30 ml/min) vs only 5 % in the DN group for the same eGFR range. Statistically however these differences were not significant .Teresa[40] et al study showed higher creatinine values in the NDN group which were significant. Dakshinamoorthy[41] et al also showed higher serum creatinine values in the NDN which were however not statistically significant.

6) BMI and cholesterol levels as risk factors

The BMI in the DN and non DN group were similar with a mean BMI of 23 in both groups .In the Teresa et al study the BMI was 24 and 25 in the DN and non DN group respectively .The mean serum cholesterol levels were higher in the DN group than the non DN group (160 mg/dl vs 138 mg/dl) .This difference was not statistically significant .

CONCORDANCE OF DIABETIC NEPHROPATHY AND RETINOPATHY

The presence of diabetic retinopathy was evaluated in each of the groups DN, non DN and the non DN superimposed on DN group .The results showed that 53 % of the patients with biopsy proven nephropathy had retinopathy but 47 % of the DN group did not have retinopathy.

Similarly 36 % patients of the non DN group had diabetic retinopathy while 64% did not . In the (NDN + DN) group 50 % had retinopathy .The sensitivity of retinopathy for nephropathy was 71.4 % with a specificity of 63.6 % .Statistical analysis by Chi Squared testing showed that retinopathy was ***not*** significant to differentiate DN from non DN (p value 0.6).

Parving[4] et al in their study published in 1992 ,showed that 41 % of their patients with biopsy proven diabetic nephropathy lacked retinopathy .According to Parving the reason for this could be hemodynamic factors ,causing impaired autoregulation and elevated glomerular hydraulic pressure this causes accelerated microangiopathy of the kidney compared to the retina .The Parving study also showed lack of retinopathy to be a poor predictor of non diabetic kidney disease .In the J Prakash[3] study published in India 2007 showed their concordance of retinopathy and biopsy proven nephropathy to be 60 %.Their study also showed 40 % NDN patients to have retinopathy as well .They further concluded in their study that in the

absence of retinopathy there was a 50 % chance of DN or non DN hence it is a poor predictor of non diabetic kidney disease .Teresa[40] et al study showed absence of retinopathy to have a sensitivity of 82 % ,specificity of 70 % and positive predictive value of 83 % in predicting non diabetic kidney disease .They further state that with absence of retinopathy and proteinuria > 2g/day taken together the positive predictive value increases to 90 % .

Thus the concordance of diabetic nephropathy with retinopathy was 53 % in our study, while in other studies it was around 60%. In the NDN group retinopathy was seen in 36 % which was comparable to other studies in which retinopathy was seen in upto 40%.

CONCLUSIONS

1. The concordance between diabetic nephropathy and retinopathy was 53 % in our study
2. There was no difference between the diabetic nephropathy and non diabetic nephropathy group in terms of duration of diabetes ,age, quantum of proteinuria, estimated GFR and hypertension.
3. The prevalence of non diabetic nephropathy was 34 % in our study.
4. Retinopathy was seen in 36 % of the non diabetic nephropathy group as well.
5. A renal biopsy is required for the accurate diagnosis of diabetic or non diabetic kidney disease in a proteinuric type 2 diabetic even in the presence of diabetic retinopathy .

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PROFORMA

Name : Age : Sex:

Occupation:

Address:

Present complaints :

Past history : Diabetes : Hypertension:

Duration of diabetes :

H/o Retinopathy:

H/o Neuropathy :

H/o Ischaemichea:

Rt Disease : H/o CVA :

Hypertension – Duration

Diabetes Treatment – OHA Insulin –

Family H/o – Diabetes Hypertension –

Personnel H/o – smoking Alcohol-

Marital status –

Physical examination –

1)Height 2)weight 3)BMI-

4)Pulse rate 5)Blood pressure - 6)peripheral pulses

Fundus examination –

Non diabetic nephropathy-

Mild NPDR – Moderate NPDR – Severe

NPDR -

Proliferative diabetic nephropathy – Macular edema –

CVS- RS- CNS –

Abdomen –

Lab analysis –

Urine analysis - protein sediment

Urine PCR - S.Creatinine - Blood sugar –FBS -PPBS

Urine c/s – USG KUB –

ECG – ECHO –

Renal biopsy -

MASTER CHART

s.no	name	nc	age	age_g	sex	bmi	bmi_g	dia_dur	dia_g	htn	htn_dur	htn_g	prot_ur	pcr	pcr_g	creat	egfr	egfr_g	chol	usg	oha	insulin	cvs	neurop	retino	npdr	pdr	ffa	prpp	systdls	act_sedi	biopsy	dia_neph	mesang	hyalinos	ifta	glom_scl	imp	imp_a	v35
1	venkataiyan	5317/13	51	51-60	Male	23	Normal	5	05-Feb	Yes	5	05-Feb	4+	6	> 5.0	6	10	<= 15	150	Cont	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
2	desappan	5437/13	73	71-80	Male	18	Under weight	5	05-Feb	Yes	5	05-Feb	3+	3.5	1.1-3.5	3.8	15	<= 15	140	Cont	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
3	asra boo	5472/13	59	51-60	Female	25	Over weight	10	10-May	Yes	10	10-May	Trace	2	1.1-3.5	4.1	15	<= 15	180	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
4	theresa	893/13	55	51-60	Female	21	Normal	3	05-Feb	Yes	1	<= 2	3+	1.4	1.1-3.5	6	10	<= 15	120	Cont	No	Yes	LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
5	parvathy	5233/13	48	41-50	Female	18	Under weight	2	<= 2	Yes	2	<= 2	1+	1.2	1.1-3.5	1.2	58	45-59	200	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
6	valli.j	5627/13	45	41-50	Female	18	Under weight	16	> 10	Yes	11	> 10	3+	7	> 5.0	8	5	<= 15	110	Cont	No	Yes	LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
7	benny d	5633/13	45	41-50	Male	20	Normal	3	05-Feb	Yes	1	<= 2	3+	6.8	> 5.0	7	11	<= 15	165	Normal	No	Yes	LVD Dysfunction	No	Yes	No	No	Yes	No	No	No	No	.	.	.	.				
8	kasi	5652/13	43	41-50	Female	21	Normal	3	05-Feb	Yes	1	<= 2	3+	5	3.6-5.0	1	70	60-89	190	Normal	No	No	LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
9	devaraj	5689/13	53	51-60	Male	16	Under weight	4	05-Feb	Yes	1	<= 2	4+	8	> 5.0	5	14	<= 15	200	Cont	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
10	mani.g	5789/13	60	51-60	Male	18	Under weight	6	10-May	Yes	1	<= 2	4+	6	> 5.0	5	14	<= 15	120	Normal	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
11	chandrasekar		78	71-80	Male	18	Under weight	5	05-Feb	Yes	1	<= 2	Trace	6	> 5.0	1.5	28	16-29	140	Normal	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	No	No	Yes	Yes	1	glob glom		
12	s.selvam	6004/13	53	51-60	Male	24	Normal	6	10-May	Yes	1	<= 2	3+	2.4	1.1-3.5	6	12	<= 15	110	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
13	sekarsamy	6100/13	63	61-70	Male	16	Under weight	1	<= 2	Yes	1	<= 2	3+	3.4	1.1-3.5	6.2	11	<= 15	110	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
14	nirmala	592/13	37	31-40	Female	20	Normal	10	10-May	Yes	0.5	<= 2	3+	8	> 5.0	1.6	45	45-59	110	Normal	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
15	samanasu		49	41-50	Male	23	Normal	2	<= 2	Yes	2	<= 2	3+	3.5	1.1-3.5	6	12	<= 15	140	Cont	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
16	sakthivel.r	6084/13	45	41-50	Male	20	Normal	25	> 10	No	2	<= 2	3+	5	3.6-5.0	4	14	<= 15	124	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
17	k.nagaraj	1409/13	57	51-60	Male	25	Over weight	15	> 10	Yes	0.5	<= 2	4+	4.3	3.6-5.0	2.4	12	<= 15	220	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
18	laksmikanth	5129/13	65	61-70	Female	23	Normal	5	05-Feb	Yes	20	> 10	1+	0.6	0.5-1.0	1.4	54	45-59	180	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
19	munusamy. m	6387/13	50	41-50	Male	24	Normal	5	05-Feb	No	0	No hypertension	3+	3.5	1.1-3.5	4	12	<= 15	180	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
20	raja.r	6642/13	48	41-50	Male	23	Normal	20	> 10	Yes	2	<= 2	1+	1.4	1.1-3.5	1.7	56	45-59	235	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
21	e.mohan	4338/13	61	61-70	Male	20	Normal	3	05-Feb	Yes	2.5	05-Feb	4+	7.1	> 5.0	2.6	20	16-29	180	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
22	s.rajendran	3401/13	50	41-50	Male	21	Normal	20	> 10	Yes	0.3	<= 2	4+	6	> 5.0	2.8	20	16-29	200	Normal	No	No	No LVD Dysfunction	No	Yes	No	No	No	Yes	Yes	No	No	.	.	.	.				
23	saravanan	1120/13	35	31-40	Male	22	Normal	8	10-May	Yes	0.3	<= 2	3+	3.9	3.6-5.0	0.9	86	60-89	140	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	Yes	No	No	No	.	.	.	.				
24	p.sampath	5110/12	54	51-60	Male	21	Normal	15	> 10	No	0	No hypertension	4+	6	> 5.0	3.6	22	16-29	160	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
25	Rangan	4888/12	54	51-60	Male	21	Normal	3	05-Feb	Yes	1	<= 2	Trace	0.4	0.5-1.0	3	20	16-29	118	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
26	gowri	3345/12	65	61-70	Female	22	Normal	3	05-Feb	No	0	No hypertension	3+	3.5	1.1-3.5	3.5	14	<= 15	158	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
27	rajendran a	3446/12	48	41-50	Male	23	Normal	3	05-Feb	No	0	No hypertension	4+	6	> 5.0	2.6	32	30-44	134	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
28	chellan	3567/12	69	61-70	Male	19	Under weight	5	05-Feb	Yes	1	<= 2	4+	8	> 5.0	2.8	22	16-29	150	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				

29	t.suguna	3447/12	41	41-50	Female	21	Normal	8	10-May	Yes	2	<= 2	4+	7	> 5.0	3.2	21	16-29	170	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
30	R.selvaraj	3657/12	53	51-60	Male	24	Normal	7	10-May	Yes	2	<= 2	3+	4	3.6-5.0	3.3	12	<= 15	119	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
31	kannamal	3552/13	57	51-60	Female	21	Normal	6	10-May	Yes	1	<= 2	4+	7	> 5.0	2.6	20	16-29	112	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
32	malliappan	3112/12	57	51-60	Male	20	Normal	5	05-Feb	No	0	No hypertension	2+	1.8	1.1-3.5	3.2	16	16-29	130	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
33	e.subramani	4107/12	48	41-50	Male	21	Normal	3	05-Feb	No	0	No hypertension	2+	2	1.1-3.5	2.2	40	30-44	118	Borderlin	No	No	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	.	.	.	.			ATN	
34	parameshwari	4213/12	51	51-60	Female	24	Normal	5	05-Feb	Yes	2	<= 2	4+	8	> 5.0	3.1	21	16-29	130	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
35	govindan	4446/12	60	51-60	Male	20	Normal	5	05-Feb	Yes	2	<= 2	4+	5	3.6-5.0	2.8	18	16-29	140	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
36	jeeva	3487/12	42	41-50	Male	22	Normal	1	<= 2	No	0	No hypertension	2+	2	1.1-3.5	1.5	48	45-59	160	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	.	.	.	.			ATN	
37	prasadu	4412/12	45	41-50	Male	21	Normal	2	<= 2	No	0	No hypertension	1+	1.8	1.1-3.5	1.6	46	45-59	128	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	Yes	No	No	No	2	yes=1	DN	
38	ramalingam	4444/12	59	51-60	Male	21	Normal	2	<= 2	No	0	No hypertension	2+	2.4	1.1-3.5	1.5	40	30-44	112	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	Yes	Yes	No	Yes	2	yes=1	DN	
39	shanmugam	3215/12	62	61-70	Male	20	Normal	3	05-Feb	Yes	1	<= 2	1+	1.8	1.1-3.5	1.5	38	30-44	130	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	.	.	.	.				
40	r.venkatesan	2134/12	58	51-60	Male	22	Normal	10	10-May	Yes	2	<= 2	4+	4.4	3.6-5.0	1.5	42	30-44	125	Normal	Yes	No	LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	Yes	Yes	Yes	Yes	No	2	no=0	DN	
41	Dharmalingam	2234/12	62	61-70	Male	22	Normal	8	10-May	Yes	4	05-Feb	4+	4.2	3.6-5.0	1.6	40	30-44	234	Normal	No	Yes	LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	Yes	Yes	Yes	Yes	2	no=0	DN	
42	lakshmanan	3110/12	68	61-70	Male	20	Normal	2	<= 2	Yes	5	05-Feb	3+	3.2	1.1-3.5	1.4	46	45-59	221	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	Yes	Yes	Yes	Yes	2	yes=1	DN	
43	Baby	3211/12	60	51-60	Female	25	Over weight	4	05-Feb	Yes	2	<= 2	3+	3.4	1.1-3.5	1.5	40	30-44	180	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
44	G.Rajasekar	4325/12	51	51-60	Male	21	Normal	15	> 10	Yes	5	05-Feb	4+	4.4	3.6-5.0	4.8	16	16-29	114	Normal	No	Yes	No LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
45	V.ponnusamy	4467/12	63	61-70	Male	22	Normal	11	> 10	Yes	5	05-Feb	1+	1	0.5-1.0	1.5	38	30-44	240	Normal	No	Yes	No LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
46	veeramani	3215/12	50	41-50	Male	22	Normal	15	> 10	Yes	4	05-Feb	4+	7	> 5.0	3.6	16	16-29	130	Borderlin	No	Yes	LVD Dysfunction	No	Yes	Yes	No	Yes	Yes	No	No	No	.	.	.	.				
47	Paneerselvam	4531/12	45	41-50	Male	21	Normal	3	05-Feb	No	0	No hypertension	1+	1.5	1.1-3.5	2	36	30-44	110	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
48	Janathanan	4512/12	45	41-50	Male	20	Normal	16	> 10	Yes	5	05-Feb	1+	1.3	1.1-3.5	2.2	35	30-44	120	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
49	k.Sigamani	1123/12	65	61-70	Male	20	Normal	2	<= 2	No	0	No hypertension	Trace	0.3	0.5-1.0	2	35	30-44	210	Borderlin	Yes	No	LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
50	k.Aswini	1128/12	52	51-60	Female	25	Over weight	3	05-Feb	No	0	No hypertension	2+	2.4	1.1-3.5	1.5	40	30-44	180	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
51	R.subramanian	4367/12	69	61-70	Male	20	Normal	9	10-May	Yes	3	05-Feb	Trace	0.6	0.5-1.0	1.7	38	30-44	130	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
52	Viji	4267/12	42	41-50	Female	24	Normal	3	05-Feb	No	0	No hypertension	1+	1.2	1.1-3.5	1.5	38	30-44	120	Normal	Yes	No	No LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
53	Sarala	3117/12	47	41-50	Female	21	Normal	15	> 10	Yes	4	05-Feb	1+	1.3	1.1-3.5	2.8	40	30-44	130	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
54	Thangavel	2212/12	60	51-60	Male	22	Normal	5	05-Feb	Yes	2	<= 2	3+	3	1.1-3.5	2	36	30-44	120	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
55	Raziyabee	3576/11	51	51-60	Female	25	Over weight	4	05-Feb	Yes	2	<= 2	1+	1.5	1.1-3.5	1.8	38	30-44	180	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				

56	Alangavam	3778/12	45	41-50	Female	21	Normal	6	10-May	Yes	2	<= 2	2+	2.3	1.1-3.5	1.7	24	16-29	210	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
57	V.Sivagami	1681/12	40	31-40	Female	23	Normal	4	05-Feb	No	0	No hypertension	2+	2.4	1.1-3.5	0.9	70	60-89	140	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
58	Balan	5060/12	54	51-60	Male	22	Normal	6	10-May	Yes	2	<= 2	3+	3.3	1.1-3.5	1.6	39	30-44	226	Normal	No	Yes	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
59	Baskar A	5167/12	48	41-50	Male	23	Normal	8	10-May	Yes	1	<= 2	4+	4.3	3.6-5.0	8	12	<= 15	130	Normal	No	Yes	No LVD Dysfunction	No	No	No	No	No	No	Yes	Yes	Yes	.	.	.	.			anti-GBM	
60	K.Velu	2187/12	44	41-50	Male	24	Normal	4	05-Feb	No	0	No hypertension	Trace	0.5	0.5-1.0	1.6	42	30-44	120	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
61	Soundarpandi	4634/12	51	51-60	Male	26	Over weight	4	05-Feb	Yes	1	<= 2	2+	2.5	1.1-3.5	2.5	21	16-29	180	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
62	P.Balachandran	5612/12	57	51-60	Male	21	Normal	4	05-Feb	No	0	No hypertension	1+	1.5	1.1-3.5	2.4	23	16-29	150	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
63	Aravindan	4523/12	55	51-60	Male	23	Normal	5	05-Feb	Yes	2	<= 2	3+	3.4	1.1-3.5	2.3	21	16-29	140	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
64	Sundramoorthy	5778/12	54	51-60	Male	24	Normal	6	10-May	Yes	1	<= 2	4+	4.4	3.6-5.0	1.5	38	30-44	132	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
65	Veeramani	5768/12	49	41-50	Male	22	Normal	3	05-Feb	Yes	1	<= 2	2+	2.2	1.1-3.5	1.4	46	45-59	146	Normal	No	Yes	No LVD Dysfunction	Yes	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
66	Sekar	750/12	47	41-50	Male	21	Normal	2	<= 2	No	0	No hypertension	4+	4.4	3.6-5.0	1.8	39	30-44	130	Normal	Yes	No	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	.	.	.	.			Memb nephropathy	
67	Kumar.D	5758/12	45	41-50	Male	25	Over weight	3	05-Feb	No	0	No hypertension	2+	2	1.1-3.5	2.2	32	30-44	224	Normal	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	Yes	.	.	.	.				
68	C.Pugazhenti	5330/12	55	51-60	Male	21	Normal	4	05-Feb	No	0	No hypertension	3+	1.6	1.1-3.5	2.2	35	30-44	120	Normal	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	Yes	Yes	Yes	No	No	Yes		yes	DN+PiGN	
69	Arumugam	5435/12	63	61-70	Male	21	Normal	5	05-Feb	No	0	No hypertension	1+	1.5	1.1-3.5	2.1	24	16-29	138	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	Yes	.	.	.	.			CIN	
70	Dhanan.V	4312/12	40	31-40	Male	22	Normal	2	<= 2	Yes	1	<= 2	2+	2	1.1-3.5	1.5	40	30-44	111	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
71	Anandh	402/13	30	21-30	Male	29	Over weight	4	05-Feb	Yes	2	<= 2	3+	3.1	1.1-3.5	2.5	22	16-29	190	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
72	Vanitha	4336/12	26	21-30	Female	21	Normal	4	05-Feb	No	0	No hypertension	3+	3.9	3.6-5.0	2	54	45-59	130	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
73	farooq ali	2456/12	50	41-50	Male	26	Over weight	3	05-Feb	Yes	1	<= 2	2+	2.2	1.1-3.5	2.3	20	16-29	240	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
74	Tirupathi rao	3425/11	55	51-60	Male	24	Normal	4	05-Feb	Yes	1	<= 2	3+	3.4	1.1-3.5	2	24	16-29	180	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
75	Laxmipathy	3521/12	52	51-60	Male	25	Over weight	3	05-Feb	No	0	No hypertension	2+	2.5	1.1-3.5	2.8	18	16-29	140	Normal	No	Yes	LVD Dysfunction	Yes	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
76	Nagaraj	2314/12	35	31-40	Male	21	Normal	2	<= 2	No	0	No hypertension	3+	3.3	1.1-3.5	1.9	38	30-44	140	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
77	R.Chellamal	2345/11	70	61-70	Female	23	Normal	4	05-Feb	No	0	No hypertension	2+	2.4	1.1-3.5	2	18	16-29	130	Borderlin	Yes	No	LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
78	K.Adhi	2134/12	62	61-70	Male	21	Normal	6	10-May	Yes	2	<= 2	4+	7	> 5.0	2.1	20	16-29	124	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
79	Latha	2133/12	30	21-30	Female	20	Normal	1	<= 2	No	0	No hypertension	4+	6	> 5.0	2	31	30-44	132	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				

80	S.Yuvarajan	2764/13	29	21-30	Male	24	Normal	1	<= 2	No	0	No hypertension	3+	3.2	1.1-3.5	2.5	20	16-29	124	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
81	V.Manuel	3191/13	31	31-40	Male	21	Normal	1	<= 2	No	0	No hypertension	1+	1	0.5-1.0	1.4	56	45-59	120	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
82	A.Ramamoorht	3196/13	64	61-70	Male	24	Normal	4	05-Feb	Yes	2	<= 2	Trace	0.6	0.5-1.0	3	16	16-29	146	Borderlin	No	No	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
83	G.Srinivasan	3061/13	50	41-50	Male	28	Over weight	5	05-Feb	Yes	3	05-Feb	1+	0.6	0.5-1.0	3.5	16	16-29	126	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
84	A.Shanmugam	3112/13	44	41-50	Male	30	Over weight	3	05-Feb	Yes	1	<= 2	3+	3.2	1.1-3.5	1.6	52	45-59	130	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
85	R.Veera	2132/12	63	61-70	Male	20	Normal	2	<= 2	Yes	2	<= 2	1+	1.6	1.1-3.5	2.2	20	16-29	110	Borderlin	Yes	No	No LVD Dysfunction	Yes	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
86	V.Veeramani	3214/12	56	51-60	Male	21	Normal	3	05-Feb	Yes	1	<= 2	4+	6	> 5.0	2.5	21	16-29	158	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
87	Amudha	2114/13	28	21-30	Female	26	Over weight	2	<= 2	Yes	1	<= 2	3+	3.2	1.1-3.5	1.5	52	45-59	130	Borderlin	Yes	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
88	Soundaram	3211/13	52	51-60	Female	23	Normal	2	<= 2	Yes	2	<= 2	3+	3.2	1.1-3.5	2.4	21	16-29	140	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
89	Masilamani	4112/13	38	31-40	Male	27	Over weight	3	05-Feb	Yes	3	05-Feb	3+	3.6	3.6-5.0	2.4	26	16-29	160	Normal	No	Yes	No LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
90	Saroja.A	3422/13	50	41-50	Female	28	Over weight	2	<= 2	Yes	2	<= 2	3+	3.3	1.1-3.5	2.8	16	16-29	120	Borderlin	No	Yes	LVD Dysfunction	Yes	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
91	Muthusamy.R	2145/13	35	31-40	Male	23	Normal	2	<= 2	Yes	2	<= 2	3+	3.4	1.1-3.5	2.5	50	45-59	152	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
92	Mohan	2988/13	50	41-50	Male	21	Normal	5	05-Feb	Yes	2	<= 2	4+	8	> 5.0	3.8	12	<= 15	140	Borderlin	No	Yes	No LVD Dysfunction	Yes	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
93	Ranganathan	3712/13	37	31-40	Male	22	Normal	4	05-Feb	Yes	2	<= 2	4+	4	3.6-5.0	1.9	40	30-44	130	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
94	Thiagrajan	4328/13	41	41-50	Male	28	Over weight	4	05-Feb	Yes	4	05-Feb	4+	8.1	> 5.0	2	40	30-44	190	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
95	Sulochana	5233/13	63	61-70	Female	21	Normal	2	<= 2	Yes	2	<= 2	2+	2.2	1.1-3.5	2.8	20	16-29	160	Borderlin	No	Yes	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
96	Rajendran	2312/13	44	41-50	Male	20	Normal	4	05-Feb	Yes	2	<= 2	3+	3.2	1.1-3.5	2.9	15	<= 15	190	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
97	S.Kasi	4512/13	60	51-60	Male	21	Normal	4	05-Feb	Yes	2	<= 2	3+	3.2	1.1-3.5	1.6	50	45-59	186	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
98	Noorjahan	5812/13	58	51-60	Female	28	Over weight	4	05-Feb	Yes	4	05-Feb	2+	2	1.1-3.5	2.2	20	16-29	149	Borderlin	No	Yes	LVD Dysfunction	Yes	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
99	Prabakaran	2314/13	60	51-60	Male	24	Normal	4	05-Feb	Yes	2	<= 2	4+	7	> 5.0	2.8	18	16-29	140	Borderlin	No	Yes	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
100	Anjammal	2134/13	51	51-60	Female	21	Normal	5	05-Feb	Yes	1	<= 2	3+	3	1.1-3.5	1.7	50	45-59	136	Borderlin	Yes	No	No LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
101	Muthusamy.R	2134/13	37	31-40	Male	22	Normal	5	05-Feb	Yes	2	<= 2	4+	4	3.6-5.0	2	40	30-44	120	Borderlin	No	Yes	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
102	A.Y Clement	4312/12	72	71-80	Male	19	Under weight	4	05-Feb	Yes	1	<= 2	3+	3.1	1.1-3.5	1.4	52	45-59	110	Normal	Yes	No	No LVD Dysfunction	Yes	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
103	K.Kumar	4112/12	53	51-60	Male	24	Normal	3	05-Feb	Yes	1	<= 2	4+	4.2	3.6-5.0	1.8	44	30-44	132	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
104	S.Ravi	2113/13	34	31-40	Male	24	Normal	2	<= 2	No	1	<= 2	4+	7	> 5.0	2.4	20	16-29	124	Normal	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
105	A.Kattaya	2074/13	60	51-60	Male	22	Normal	3	05-Feb	Yes	1	<= 2	4+	4	3.6-5.0	2	25	16-29	220	Normal	Yes	No	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
106	Seetha	1494/13	60	51-60	Female	21	Normal	4	05-Feb	Yes	2	<= 2	4+	6.4	> 5.0	2	20	16-29	118	Borderlin	Yes	No	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
107	Pregalathan	4147/13	45	41-50	Male	26	Over weight	3	05-Feb	Yes	1	<= 2	4+	4	3.6-5.0	3.8	16	16-29	148	Borderlin	No	Yes	No LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
108	G.Rajasekar	306/13	51	51-60	Male	25	Over weight	15	> 10	Yes	14	> 10	4+	4	3.6-5.0	3	22	16-29	154	Borderlin	No	Yes	LVD Dysfunction	Yes	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
109	K.Mani	2836/11	52	51-60	Male	21	Normal	5	05-Feb	Yes	5	05-Feb	1+	1.6	1.1-3.5	1.4	45	45-59	150	Normal	Yes	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
110	Gowri.M	1695/13	67	61-70	Female	18	Under weight	25	> 10	Yes	6	10-May	1+	1.4	1.1-3.5	2.2	22	16-29	140	Normal	No	Yes	LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				

111	Shanti.m	1595/08	38	31-40	Female	21	Normal	3	05-Feb	Yes	2	<= 2	4+	4	3.6-5.0	1.4	50	45-59	130	Normal	Yes	No	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	.	.	.	.			MCD/DMP	
112	Kamalaveni.R	2314/10	37	31-40	Female	19	Under weight	2	<= 2	No	0	No hypertension	2+	2.3	1.1-3.5	3	18	16-29	120	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
113	K.Pongajavalli	2915/13	56	51-60	Female	21	Normal	10	10-May	Yes	6	10-May	Trace	1	0.5-1.0	2.5	24	16-29	130	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
114	Pichai	2865/13	72	71-80	Male	20	Normal	3	05-Feb	Yes	1	<= 2	3+	3.8	3.6-5.0	1.7	42	30-44	128	Normal	No	Yes	No LVD Dysfunction	Yes	No	No	No	No	No	No	No	Yes	Yes	Yes	No	Yes	2	yes 6/8	Diab Neph	
115	Rathinam	1234/12	70	61-70	Male	21	Normal	6	10-May	Yes	15	> 10	2+	1.2	1.1-3.5	1.5	45	45-59	136	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	Yes	Yes	No	Yes	2	yes 2/4	Diab Neph	
116	Rajan leo	2246/10	55	51-60	Male	24	Normal	15	> 10	No	0	No hypertension	1+	1.1	1.1-3.5	1.3	58	45-59	130	Normal	No	Yes	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
117	Arumugam	399/13	60	51-60	Male	20	Normal	3	05-Feb	Yes	3	05-Feb	3+	3.1	1.1-3.5	2	26	16-29	140	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
118	Rajendran	294/13	44	41-50	Male	23	Normal	1	<= 2	Yes	1	<= 2	3+	3	1.1-3.5	3.8	18	16-29	130	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
119	Chandrasekaran	4535/13	51	51-60	Male	21	Normal	2	<= 2	No	0	No hypertension	4+	6	> 5.0	1.4	48	45-59	240	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
120	Ramachandran	4674/13	50	41-50	Male	19	Under weight	6	10-May	No	0	No hypertension	4+	6.5	> 5.0	4	16	16-29	132	Borderlin	No	No	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
121	Ayub basha	4813/13	32	31-40	Male	26	Over weight	3	05-Feb	No	0	No hypertension	4+	5	3.6-5.0	2.1	44	30-44	226	Borderlin	Yes	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
122	Bala.s	5024/13	34	31-40	Male	22	Normal	3	05-Feb	No	0	No hypertension	4+	7	> 5.0	3	20	16-29	132	Borderlin	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
123	Ayesha.M	5140/13	48	41-50	Female	25	Over weight	4	05-Feb	No	0	No hypertension	3+	3.8	3.6-5.0	4	15	<= 15	120	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
124	R.Subbamal	5374/13	55	51-60	Female	21	Normal	1	<= 2	No	0	No hypertension	3+	3.4	1.1-3.5	4	14	<= 15	130	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
125	K.Selvaraj	5380/13	43	41-50	Male	25	Over weight	3	05-Feb	Yes	1	<= 2	1+	0.8	0.5-1.0	4.4	12	<= 15	120	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
126	C.Jeeva	5144/13	44	41-50	Male	23	Normal	2	<= 2	Yes	1	<= 2	4+	5.3	> 5.0	1.6	44	30-44	156	Borderlin	Yes	No	No LVD Dysfunction	Yes	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
127	Balaraman	4224/12	55	51-60	Male	22	Normal	2	<= 2	Yes	2	<= 2	2+	1.9	1.1-3.5	4	12	<= 15	124	Borderlin	No	No	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
128	Soundarajan	4227/12	54	51-60	Male	21	Normal	3	05-Feb	Yes	2	<= 2	4+	7	> 5.0	4	12	<= 15	154	Borderlin	No	Yes	No LVD Dysfunction	Yes	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
129	Logabalan	4381/13	65	61-70	Male	22	Normal	5	05-Feb	No	0	No hypertension	4+	7.4	> 5.0	4.4	10	<= 15	120	Borderlin	No	No	LVD Dysfunction	Yes	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
130	Nagan	3402/13	56	51-60	Male	21	Normal	3	05-Feb	Yes	3	05-Feb	4+	7.8	> 5.0	3	15	<= 15	186	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
131	V.Gunasekar	4235/13	50	41-50	Male	22	Normal	2	<= 2	Yes	1	<= 2	4+	5	3.6-5.0	4.5	16	16-29	210	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
132	A.Mohan	4144/13	55	51-60	Male	21	Normal	1	<= 2	Yes	1	<= 2	Trace	0.5	0.5-1.0	3	18	16-29	128	Normal	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
133	Pregalathan	4147/13	44	41-50	Male	22	Normal	2	<= 2	Yes	1	<= 2	4+	4.2	3.6-5.0	4.2	15	<= 15	130	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
134	Selvam	4189/13	51	51-60	Male	23	Normal	4	05-Feb	Yes	2	<= 2	4+	5.4	> 5.0	4.5	15	<= 15	128	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
135	Andiyappan	4628/13	58	51-60	Male	21	Normal	5	05-Feb	Yes	2	<= 2	2+	2.5	1.1-3.5	3.8	17	16-29	130	Borderlin	No	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
136	S.Rajendran	3401/13	50	41-50	Male	26	Over weight	20	> 10	Yes	1	<= 2	4+	6.1	> 5.0	2.6	40	30-44	220	Normal	No	Yes	LVD Dysfunction	Yes	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
137	Elango	233/13	65	61-70	Male	22	Normal	5	05-Feb	Yes	1	<= 2	4+	5	3.6-5.0	2.6	38	30-44	130	Normal	No	Yes	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	Yes	Yes	No	Yes	2	yes 4/10	Diab Neph	

138	Parvathy	122/13	48	41-50	Female	20	Normal	6	10-May	Yes	1	<= 2	2+	1.5	1.1-3.5	1.4	56	45-59	120	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	Yes	Yes	No	Yes	<10 %	yes 2/15	Diab Neph	
139	A.L Kannan	5177/13	64	61-70	Male	20	Normal	0	<= 2	No	0	No hypertension	4+	4.4	3.6-5.0	3	16	16-29	130	Normal	No	No	No LVD Dysfunction	No	Yes	Yes	No	No	No	No	No	No	.	.	.	.				
140	Baskar.M	1734/12	56	51-60	Male	26	Over weight	3	05-Feb	Yes	1	<= 2	Trace	0.6	0.5-1.0	2.2	23	16-29	146	Borderlin	No	Yes	No LVD Dysfunction	No	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
141	Mani.M	5975/13	50	41-50	Male	24	Normal	20	> 10	Yes	5	05-Feb	4+	4.1	3.6-5.0	3.6	16	16-29	140	Normal	No	Yes	LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	No	.	.	.	.				
142	Arasi	2908/12	65	61-70	Female	21	Normal	21	> 10	Yes	2	<= 2	4+	6.8	> 5.0	2.6	20	16-29	124	Borderlin	No	Yes	No LVD Dysfunction	Yes	Yes	No	No	Yes	Yes	No	No	No	.	.	.	.				
143	Desappan	2130/13	50	41-50	Male	23	Normal	3	05-Feb	Yes	1	<= 2	4+	4.5	3.6-5.0	1.3	48	45-59	186	Normal	Yes	No	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	No	No	No	Yes	<10%	yes 1/23	mcd/fsgs	neg
144	Devadoss	2210/13	61	61-70	Male	21	Normal	7	10-May	Yes	2	<= 2	2+	2.4	1.1-3.5	1.5	46	45-59	150	Normal	No	Yes	No LVD Dysfunction	No	Yes	Yes	No	Yes	No	No	No	Yes	Yes	No	No	Yes	10-15%	yes 16/21	Diab Neph	neg
145	Nagammal	1230/13	63	61-70	Female	22	Normal	25	> 10	Yes	15	> 10	4+	5.5	> 5.0	1.6	40	30-44	126	Normal	No	Yes	No LVD Dysfunction	No	No	No	No	No	No	Yes	Yes	Yes	No	Yes	No	Yes	10.00%	yes 2/8	DN +Hsp	IgA 3+
146	Rathinam	333/12	70	61-70	Male	18	Under weight	6	10-May	Yes	10	10-May	2+	1.5	1.1-3.5	1.5	45	45-59	110	Normal	No	Yes	No LVD Dysfunction	Yes	Yes	Yes	No	Yes	No	No	No	Yes	Yes	Yes	No	Yes	2	yes 2/4	DN	neg
147	Saravanan	546/12	36	31-40	Male	24	Normal	3	05-Feb	No	0	No hypertension	3+	3.2	1.1-3.5	1.5	58	45-59	132	Normal	Yes	No	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	Yes	Yes	Yes	Yes	50.00%	yes 4/7	DN	neg
148	Jaya	6412/12	64	61-70	Female	22	Normal	3	05-Feb	Yes	1	<= 2	3+	2.2	1.1-3.5	1.5	42	30-44	122	Normal	Yes	No	No LVD Dysfunction	Yes	No	No	No	No	No	No	No	Yes	No	Yes	No	No	10.00%	yes ,2/13	Mem Neph	IgG(3+),c3(3+)
149	Saravanan.M	232/11	36	31-40	Male	22	Normal	3	05-Feb	Yes	3	05-Feb	3+	3.3	1.1-3.5	1.9	42	30-44	188	Normal	No	Yes	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	Yes	Yes	No	Yes	yes,40%	yes,12/12	Diab glomscle	neg
150	Devdoss	2123/12	61	61-70	Male	21	Normal	4	05-Feb	Yes	2	<= 2	4+	4.4	3.6-5.0	1.5	40	30-44	210	Normal	No	Yes	No LVD Dysfunction	Yes	No	No	No	No	No	No	No	Yes	Yes	Yes	No	Yes	10-15%	yes,16/21	DN	neg
151	Jeyaprakash	2312/11	52	51-60	Male	24	Normal	7	10-May	No	0	No hypertension	1+	1.5	1.1-3.5	1.6	44	30-44	224	Normal	Yes	No	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	Yes	Yes	Yes	Yes	10-15%	yes 4/6	DN+nodular gloms	neg
152	Mallika Begum	1123/12	62	61-70	Female	21	Normal	15	> 10	No	0	No hypertension	1+	1.8	1.1-3.5	3	30	30-44	134	Normal	No	Yes	LVD Dysfunction	Yes	No	No	No	No	No	No	No	Yes	Yes	Yes	No	Yes	20.00%	no	DN+nodular gloms	neg
153	Mathialagan	112/14	53	51-60	Male	24	Normal	5	05-Feb	No	0	No hypertension	4+	4.2	3.6-5.0	6	15	<= 15	210	Normal	No	Yes	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	Yes	Yes	No	Yes	40.00%	yes,4/7	DN+nodular gloms	c3,Igm
154	Pitchaiya	123/13	57	51-60	Male	21	Normal	2	<= 2	No	0	No hypertension	3+	3.5	1.1-3.5	1.4	40	30-44	134	Normal	Yes	No	No LVD Dysfunction	No	No	No	No	No	No	No	No	Yes	No	No	No	No	20.00%	yes,7/23	Glomerusclerosis	neg
155	Sivanathan	2312/13	60	51-60	Male	22	Normal	4	05-Feb	Yes	2	<= 2	4+	6.2	> 5.0	4	16	16-29	124	Normal	No	No	No LVD Dysfunction	Yes	Yes	Yes	No	No	No	No	Yes	Yes	Yes	Yes	No	Yes	25.00%	yes,3/9	DN+nodular gloms	IgG(3+),c3(3+)
156	Duraisamy	234/13	58	51-60	Male	23	Normal	3	05-Feb	Yes	2	<= 2	3+	3.5	1.1-3.5	4	16	16-29	134	Normal	No	No	No LVD Dysfunction	No	No	No	No	No	No	No	Yes	Yes	No	No	No	No	2	yes,14/23	cresenteric GN	neg
157	Kumar.M	123/12	50	41-50	Male	21	Normal	4	05-Feb	Yes	2	<= 2	3+	3.4	1.1-3.5	1.4	45	45-59	224	Normal	No	No	No LVD Dysfunction	No	No	No	No	No	No	No	Yes	Yes	Yes	Yes	Yes	Yes	20.00%	no	DN+nodular gloms	neg

CONSENT FORM

TITLE OF PROJECT:

"The concordance between Diabetic Nephropathy and Retinopathy in Type 2 Diabetes mellitus."

Name of Researcher: Dr. Andrew Deepak Rajiv

Please tick to confirm

I confirm that I have read and understand the information provided to me for the above study.

I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.

I agree to take part in the above research study.

_____	_____	_____
Name of Patient	Date	Signature
_____	_____	_____
Name of Person taking consent (if different from researcher)	Date	Signature
_____	_____	_____
Researcher	Date	Signature